DISEASES

OF

THE NERVOUS SYSTEM

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THEIR PREVALENCE AND PATHOLOGY

BY

JULIUS ALTHAUS, M.D., M.R.C.P. LOND.

SENIOR PHYSICIAN TO THE HOSPITAL FOR EPILEPSY AND PARALYSIS, REGENT'S PARK; FELLOW OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY, OF THE STATISTICAL SOCIETY, AND OF THE MEDICAL SOCIETY OF LONDON; MEMBER OF THE CLINICAL SOCIETY; CORRESPONDING MEMBER OF THE SOCIÉTÉ D'HYDROLOGIE MÉDICALE DE PARIS, OF THE ELECTRO-THERAPEUTICAL SOCIETY OF NEW YORK, ETC. ETC. ETC.

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PREFACE.

In this volume I have endeavoured to elucidate the part played by diseases of the Nervous System in national pathology, and to show the laws to which their occurrence and fatality are subject. I have also fully entered into the special pathology of the several diseases of the nervous centres, which, although much remains to be done, has made gigantic strides in our time.

The pathology of peripheral nerve-diseases, and the diagnosis, prognosis, and treatment of the entire class of these maladies, will be considered in a subsequent volume.

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DISEASES

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CHAPTER I.

INTRODUCTION—CLASSIFICATION—PATHOLOGICAL PHYSIOLOGY OF THE NERVOUS CENTRES.

ONLY vague notions are at the present time entertained by the profession on the prevalence and fatality of nervous diseases; for although some able investigations into the causation of a few special affections of the nervous system have been made, and in this country more particularly by Sir George Burrows, Dr. Russell Reynolds, and Dr. Sieveking, who have endeavoured to elucidate the causes of apoplexy and epilepsy, yet nothing comprehensive has as yet been established with regard to the etiology of the entire class of these maladies. The frequency with which they occur, the place they occupy in the general mortality of the nation, the age at which they are most murderous, and the peculiar influence of sex upon their development are, as yet, quite unknown. Amongst the general public and a portion of the profession a notion is prevalent that nervous diseases, and more particularly paralysis and insanity, are, and have been for some time past, on the increase; that, inasmuch as the struggle of life is keener, the competition more intense, the work heavier, and the anxiety more absorbing, than used to be the case in previous periods of the history of mankind,

more especially in the large centres of population, the nervous system is more liable to break down by exhaustion and disease than was the case in former times. It should, however, be understood that such notions and assertions have at present no scientific base at all; but that they are made at random, and often only in order to prove or to illustrate some preconceived ideas of those who propound them. Do nervous diseases occur in a certain definite proportion, or do they vary perceptibly from time to time? Are paralysis and insanity really more frequent nowadays than formerly? Are large towns more suitable for their development than the country? Is the Anglo-Saxon race more liable to their invasion than other races? These, and a number of similar questions, still await solution, and they are not only medically, but socially speaking, of the highest importance.

That there should be so much uncertainty concerning these interesting subjects is unquestionably, to a great extent, owing to the fact that the few observers who have occupied themselves with the study of these problems have deduced their conclusions almost entirely from their own private and hospital practice; and their statements must, on account of the smallness of the numbers from which they have been derived, appear unsatisfactory and unconvincing. Even where such researches have extended over a series of years, erroneous conclusions have been arrived at. Thus M. Falret, for many years physician to La Salpétrière, ascertained that of 2,297 cases of apoplexy, 1,660 occurred in males, and only 637 in females; and Dr. Hammond, of New York, found the proportion to be 153 male, and 76 female cases in a total of 229. These numbers have led those authors to confidently express the opinion that apoplexy or cerebral hæmorrhage is more common amongst men than amongst women; and yet it is shown by my researches, which comprise not 229 as Dr. Hammond's, or 2,000 like M. Falret's, but nearly a quarter of a million cases, that women, in England at least, are rather more liable to die of apoplexy than men, and this not only for a year or two, but absolutely in the whole period over which the investigation extends. Unless we, therefore, assume that deaths from apoplexy occur in France and in the United States in a very different proportion from what they

do in England, we are led to the conclusion that the two physicians just named accidentally happened to come across more cases of males than females in the limited field of observation which was at their disposal; and that their deductions must therefore be devoid of value.

In order to arrive at truly reliable and definite results, I have addressed myself to that large storehouse of facts—the Annual Reports of the Registrar-General on disease and death in England and Wales. Dry and repulsive as large volumes filled with numbers are to the uninitiated, their contents become of surpassing interest to those who wish to recognise some general law to account for the apparently unconnected and accidental facts which come under our observation in daily life, and to perceive something fixed and stable where without such research there would appear to be nothing but chance. The only regret I have experienced in studying these volumes, which have been compiled with so much industry and care, was that they are not more complete and comprehensive, and that certain important omissions from the data placed at the disposal of the Registrar-General prevent us from gaining such a complete insight into the more hidden springs of morbid action as we would desire to obtain. But, taken as they are, they were to me of the very greatest value, as they enabled me to deal, not with hundreds or thousands, but with hundreds of thousands of cases, extending not only over a few years, but over a considerable period in the life of the nation.

Before attempting to utilise these reports, I had carefully to consider what reliance could be placed upon reports which are compiled from the certificates of death filled up by the entire body of the medical practitioners of the kingdom. It is certain that the entries of recent date are more correct than those which were made forty years ago; and the first period of five years which is included in my investigations differs in such striking respects from the subsequent ones as to create suspicions of incorrect registration. This is not surprising if we consider that the diagnosis of disease is at present not only altogether more advanced, but also much more universally disseminated amongst the general body of practitioners than formerly. Amongst many proofs for this I will only mention

that the entries of *old age as a cause of death* have gradually diminished from ten per cent. of the entire mortality in 1838 to five per cent. in 1872. Now it seems impossible to assume that thirty-five years ago twice as many people should really have died of old age simply, without disease, than is now the case; for this would show a great deterioration in the public health, which we know from other sources to have improved rather than become worse; and to my mind these numbers simply prove that formerly old age was more frequently and indiscriminately put down as a cause of death than now, when the search for disease is keener, and consequently more successful. Fortunately, however, the groups of nervous diseases which appear in the Registrar-General's reports have such striking and characteristic features that, on the whole, the errors under this head cannot amount to anything very considerable, with the exception of the first few years.

The general results of my researches on the entire class of nervous diseases may be summed up as follows:—

First Proposition.

The rate at which diseases of the nervous system prove fatal to the population of this country is a steady one, and subject to a definite law, to which there are not any or only apparent exceptions. This rate does not appear to vary perceptibly from time to time, and amounts to about twelve per cent. of the entire mortality from all causes.

This proportion is proved by the facts given in the annexed table, which shows at a glance the deaths that have been registered in England and Wales from 1838 to 1871, with the exception of the four years from 1843 to 1846, for which there is no information.

The number of years actually noted amounts therefore to thirty, which I have tabulated, not singly, which might have been confusing, but by periods of five years, or lustra, which affords a clearer and easier survey over the whole. Side by side with the numbers of deaths which took place from nervous diseases during these six lustra, I have placed the numbers of

TABLE I.

SHOWING THE DEATHS FROM NERVOUS DISEASES IN ENGLAND AND WALES, AND THEIR PROPORTION TO POPULATION AND GENERAL MORTALITY.

	Deaths from Nervous Diseases.	Population.	Death rate of N.D. in 10,000.	Deaths from all Causes.	Percentage.
1838-42	209,923	78,601,866	**26**	1,734,561	**12·10**
1843-46	Vacat.	...	...	...	...
1847 51	239,881	87,773,002	**27**	2,025,647	**11·84**
1852-56	258,221	93,085,296	**28**	2,082,346	**12·40**
1857-61	271,997	98,436,535	**27**	2,168,087	**12·54**
1862-66	293,175	103,863,878	**28**	2,397,532	**12·23**
1867-71	308,900	110,232,620	**28**	2,476,733	**12·48**

Mean Percentage of six Lustra 12·26.

the population of the kingdom, and the general mortality from all causes during the same period; for if only the deaths from nervous maladies had been tabulated, an erroneous impression that they had considerably increased might have been conveyed. Indeed, the table shows that in the first lustrum above 200,000, and in the last above 300,000, deaths occurred from these diseases; and that during the intervening lustra a steady and gradual rise in the numbers had taken place. But an actual increase of such mortality could only be taken as proved thereby if the population of the country had remained stationary during that period.

It is well known that the population of France has remained more or less stationary for a number of years past; but in this country the number of inhabitants has steadily risen, and there were in 1871 seven millions more people than there were in 1838. It was therefore necessary to calculate the death-rate for nervous diseases according to the number of inhabitants; and Table I. shows that this has varied from 26 to 28 for 10,000 people during six lustra, which gives a tolerably steady rate at which nervous diseases prove fatal to the English nation. Such a mode of calculation, however, does not give quite as accurate results as may be obtained on comparing the mortality from nervous diseases with the general mortality from all causes, as is done in the further portions of the table.

A census takes place only once in every ten years, and the population of the intervening years has therefore to be found by computation, which can never be absolutely correct. Computation cannot, for instance, take into proper account the incidental agencies of emigration and immigration, which we know to be liable to considerable fluctuations, as they are greatly dependent upon certain political and social circumstances, which, in the nature of things, are constantly changing.

If, on the contrary, we compare the deaths from nervous diseases with the deaths from all causes, we deal only with a number of separate facts, each of which has been definitely ascertained, and from which computation is altogether excluded. It is therefore evident that this latter mode of calculation offers considerable advantages over the former one.

Now, the percentages registered on Table I. are seen to be

remarkably steady and uniform, the mean average for thirty years being 12·26, and the greatest discrepancy from the mean being 0·42—that is, less than one-half per cent. for six lustra. These numbers show that the proportion in which nervous diseases prove destructive of human life is a very definite one, and subject to a certain law from which there are not any, or only apparent, deviations. Admitting that there is such a law, we could not expect that the numbers of deaths from these complaints should be absolutely identical from one lustrum to another in twenty millions of people, because there is always a variety of modifying influences at work which have to be taken into consideration. In a nation which peoples almost all the habitable regions of the globe, and is, as it were, constantly on the move, both outward and homeward, the habitual operation of such a law must always to some slight extent be interfered with. Thus, men suffering from nervous diseases come home from the colonies either for medical advice or from a wish to die in their native land; or they leave this country in search of health abroad, where perhaps they find a grave.

Again, zymotic diseases, which occur not at a steady rate, but in waves, interfere with the operation of this law, inasmuch as they carry off persons who might, but for their intercession, have died of nervous affections; and, according to the degree of intensity which they assume, these diseases either swell or reduce the entire mortality of the period. A remarkable instance of this is given by the cholera epidemic of 1849, which altered for that year the habitual death-rate of all other diseases, and particularly that of nervous maladies. In that year the habitual death-rate from these complaints reached an absolute minimum of 10·80, while it was 11·64 in the year before, and 12·88 in the year after. This interference of Asiatic cholera in 1849 caused the death-rate of the second lustrum of the table to be the lowest of all: and but for this accidental disturbance the mortality from nervous diseases would have shown even greater uniformity than is actually the case; for if we leave out the second lustrum as an abnormal one, the mean average for the remaining five becomes 12·35, and the greatest discrepancy from the mean is reduced from 0·42 to 0·25 per cent.

A final reason why the proportion of deaths from nervous diseases must vary to a slight extent is found in the vicissitudes of registration. Even if we allow for a high degree of skilled diagnosis and conscientiousness in filling up the certificates on the part of the medical practitioners of the country, there may, in a number of cases, be different views as to the complaint which actually gave rise to death. Thus, paralysis is not unfrequently complicated with Bright's disease and dropsy; and one practitioner might register the former, while another would register the latter, as the cause of death. Bronchitis will sometimes carry off persons after an attack of cerebral hæmorrhage, and either one or the other of these complaints may then be registered. There is reason to believe that during the first few years in which registration of the causes of death took place, such vicissitudes have been particularly active; and it is an interesting circumstance that if we leave out not only the second lustrum on account of its having been disturbed by cholera, but also the first for faults in registration, the death-rate for nervous diseases becomes still more uniform, as for the twenty years from 1852 to 1871 the greatest discrepancy is found to be only 0·18, instead of 0·25 for five, and 0·42 for the six lustra. After taking into account the influence of these and other circumstances combined, it becomes quite evident that slight variations in the registered mortality from nervous affections cannot alter the general law, which decrees that a certain definite proportion of the population of the kingdom—viz., about 12 per cent. of those who die—is annually cut off by nervous diseases.

The common assertion, that nervous diseases have considerably increased during the last decennia, is therefore shown to be incorrect.

Second Proposition.

Diseases of the nervous system occupy the fourth rank amongst the maladies destructive of human life, being only surpassed in fatality by zymotic, tubercular, and respiratory diseases.

This proposition is derived from the annexed table, which

TABLE II.

SHOWING THE COMPARATIVE MORTALITY FROM ZYMOTIC, TUBERCULAR, NERVOUS AND RESPIRATORY DISEASES.

	Zymotic Diseases.	Per-centage.	Tubercular Diseases.	Per-centage.	Nervous Diseases.	Per-centage.	Respiratory Diseases.	Per-centage.
1838-42	347,158	**20·04**	347,738	**20 08**	209,923	**12·10**	148,084	**8·54**
1843-46	Vacat.	...	...	...	...	...	...	...
1847-51	498,532	**24·63**	313,651	**15·50**	239,881	**11·84**	233,045	**11·50**
1852-56	454,231	**21·85**	335,275	**16·10**	258,221	**12·40**	272,970	**13·15**
1857-61	471,865	**21·80**	331,181	**15·29**	271,997	**12·54**	316,407	**14·59**
1862-66	560,015	**23·36**	347,644	**14·51**	293,175	**12·23**	357,422	**14·92**
1867-71	562,487	**22·72**	350,719	**14·16**	308,900	**12·48**	379,694	**15·33**
Mean Percentage of six Lustra . . .		**22·90**		**15·94**		**12 26**		**13·00**

shows the numbers of the population that have been carried off during six lustra by the four most important classes of maladies which appear in the Registrar-General's Reports, viz., zymotic, tubercular, nervous, and respiratory diseases. All the other classes—viz., old age, diseases of the organs of digestion, circulation, locomotion, and integumentary system, child-birth, premature birth and debility, atrophy, diseases of the genito-urinary organs, and external causes — furnish much lower quota to the general mortality, and it therefore did not appear to me worth while to include them in the list.

It is seen from this table that *zymotic diseases*, under which head the Registrar-General comprises the eruptive fevers, whooping-cough, croup, thrush, diarrhœa and dysentery, cholera, influenza, purpura and scurvy, ague, remittent and infantile fevers, typhus, typhoid, puerperal, and rheumatic fevers, erysipelas, syphilis, noma, and hydrophobia, *rank first amongst the agencies destructive of human life*, and that their fatality is subject to considerable fluctuations. The highest point was reached in 1849, when Asiatic cholera ravaged these shores. The number of deaths from these diseases amounted to 137,769 in that year, and they contributed not less than 30 per cent. to the general mortality. The mortality of the entire second lustrum amounted to 24·63, and this proportion has never again been attained. The mean average of the mortality from zymotic diseases for six lustra is seen to be 22·90—that is, more than 10 per cent. higher than that for nervous diseases.

The next great class—viz., *tubercular affections*—comprise in the Registrar-General's Reports scrofula, tabes mesenterica, phthisis, and hydrocephalus. The mortality from these distempers has not been nearly so steady as that from nervous diseases, for they are seen to have considerably diminished of late years. The difference between the first and second lustrum is indeed so great that we are led to suspect errors of registration, certain affections of the respiratory organs having probably, when registration was first commenced, been put down as tubercular, while they were later on registered under the heading of respiratory diseases. This supposition derives considerable support from the circumstance that a rise, almost approaching in sudden-

ness to the fall of tubercular diseases, is seen to have taken place from the first to the second lustrum in the class of respiratory diseases. Nevertheless, it is quite evident from the table that tubercular affections have steadily decreased of late years; and this is probably in a great measure owing to the increased prosperity of the nation, and perhaps also to some extent to improved modes of treating these diseases. Tubercular affections are less destructive than the zymotic, the mean being 15·94 against 22·90; but more so than nervous diseases, the mean of which is 12·26.

Diseases of the respiratory organs are seen to be decidedly on the increase, even if we allow a certain margin for errors in registration; and as amongst these diseases bronchitis has particularly increased, the great rise of the manufacturing industry in the country has probably been the cause of it. Bronchitis is chiefly produced by the inhalation of mechanical irritants contained in the atmosphere; and while new manufactures are constantly springing up, only little has been done to remedy the evils of smoky towns, dusty workshops, and badly-ventilated mines. Thus it has come to pass that, while in the first lustrum respiratory diseases occupied the fourth place amongst destructive distempers, they have in the present time risen to the second rank, the percentage of the last lustrum being 15·33, against 14·16 for tubercular, and 12·48 for nervous affections.

Diseases of the nervous system are therefore seen to occupy the fourth rank amongst the maladies destructive of human life.

Third Proposition.

Nervous diseases are not, as is commonly asserted, more frequent, but, on the contrary, less numerous, in large towns than in the country, and it is probable that their occurrence is powerfully influenced by race.

This proposition is derived from the annexed table, in which I have collected and compared the deaths from these diseases as they occurred respectively in London, the South-Western Counties, and Wales for a quarter of a century. The commonly

TABLE III.

SHOWING THE FATALITY OF NERVOUS DISEASES IN LONDON, S.W. COUNTIES, AND WALES.

	LONDON.			SOUTH-WESTERN COUNTIES.			WALES.		
	Deaths from Nervous Diseases.	From all Causes.	Per-centage.	Deaths from Nervous Diseases.	From all Causes.	Per-centage.	Deaths from Nervous Diseases.	From all Causes.	Per-centage.
1847-51	31,163	292,013	**10·67**	18,701	181,572	**10·32**	18,007	127,731	**14·11**
1852-56	32,379	307,620	**10·10**	21,087	175,793	**11·17**	20,564	130,675	**15·77**
1857-61	33,038	312,616	**11·05**	20,246	182,434	**11·12**	21,889	138,643	**15·80**
1862-66	38,695	370,653	**10·36**	21,460	188,993	**11·40**	23,370	150,670	**15·46**
1867-71	42,344	380,868	**11·12**	22,077	184,428	**11·99**	23,045	146,308	**15·76**
Mean Percentage of five Lustra			**10·66**			**11·20**			**15·38**

received notion that diseases of the nervous system are more prevalent and fatal in the great centres of social, professional, and commercial life than in rural districts is thereby proved to be fallacious, for it appears that the death-rate is lowest in London—viz., 10·66 per cent. of the mortality from all causes for the five lustra; that in the South-Western Counties it is higher than in London, amounting in the average to 11·20; and that it is very much higher in Wales—viz., 15·38—that is, nearly 5 per cent. more than in London.

These facts, which stand out with great clearness from Table III., appear to me eminently suggestive.

That the nervous system should be more liable to break down in the fine and wholesome atmosphere of agricultural districts than in the close and foul air of the courts and alleys which abound in great cities, seems to show that *excess of manual labour is more exhaustive to the nervous system than excess of mental labour*, and that the more nourishing and substantial food which is enjoyed by even the poorest classes in London, as compared with their brethren in the country, more than compensates them in this respect for the advantages the country affords, as far as air and climate and the supposed wholesomeness of rural pursuits are concerned.

The liability to nervous diseases would seem to diminish *pari passu* with the increased density of the population. In the years from 1841 to 1860 the density of population in London was 0·35 acres to a person; in the South-Western Counties 2·78 acres; and in Monmouthshire and Wales 4·40 acres to a person. Respiratory diseases are in this respect altogether different, being proportionately much more numerous in London than in country districts, and particularly in Wales.

I do not, however, consider that a lesser density of population explains altogether the extraordinary difference in the mortality from nervous diseases which is shown to exist between London and the South-Western Counties on one, and Wales on the other side; for I have found that Wales exceeds all English counties so strikingly in this respect, that neither density of population, nor climate, nor difference of occupation will account for that circumstance. I am therefore inclined to attribute this difference to another influence which distinguishes

Wales fundamentally from all other English counties, and that is —*difference of race.* I think we may fairly conclude, from the facts which I have brought forward, that the nervous system of the Anglo-Saxon race has greater powers of endurance and resistance to unfavourable influences than that of the Celtic race, and is better able to bear rude shocks, as well as the ordinary wear and tear of life, with impunity. If this were definitely proved by further researches, more especially concerning the French, Italians, and Spaniards, as opposed to the English, Americans, and Germans, an interesting clue would be furnished to the problem why it is that the Anglo-Saxon race appears destined to rule the world.

Fourth Proposition.

Sex has a powerful influence on the production of nervous diseases; for although in this country the population of females exceeds that of males, the deaths of males from nervous affections preponderate constantly over those of females, the male death-rate being 12·94 *and the female* 11·62 *per cent.*

This proposition is deduced from the annexed Table IV., which shows that the percentages for the deaths of males and females have been very uniform during the last twenty-five years. For *males* the highest point was 13·08, and this number actually occurs twice—viz., in the third and fifth lustra. The lowest point is 12·62, and the mean average 12·94. The highest discrepancy from the mean is 0·32; but if we exclude the first lustrum, in which the influence of cholera disturbed the ordinary death-rate, the mean average becomes 13·02, and the greatest discrepancy is reduced to 0·12.

For *females* the numbers are seen to be constantly lower than for males, but likewise very uniform. The highest point reached was 11·90, the lowest 11·07, and the mean average for the five lustra amounted to 11·62. The greatest discrepancy amounted to 0·55; but if we exclude the first lustrum for the reason stated above, the mean average becomes 11·76, and the greatest discrepancy falls to 0·14.

It is therefore proved that *males are constantly more liable*

TABLE IV.

SHOWING THE INFLUENCE OF **SEX** ON THE MORTALITY FROM DISEASES OF THE NERVOUS SYSTEM IN ENGLAND AND WALES.

	MALES.			FEMALES.		
	Deaths from Nervous Diseases.	Deaths from all Causes.	Per-centage.	Deaths from Nervous Diseases.	Deaths from all Causes.	Per-centage.
1847-51	129,196	1,023,926	**12·62**	110,684	1,000,421	**11·07**
1852-56	138,843	1,059,646	**13·02**	119,378	1,022,700	**11·67**
1857-61	145,413	1,100,671	**13·08**	126,384	1,067,416	**11 84**
1862-66	158,080	1,227,064	**12·90**	136,095	1,190,468	**11·63**
1867-71	165,591	1,275,707	**13·08**	142,809	1,201,026	**11·90**
Average Percentage of five Lustra			**12·94**			**11·62**

to die of nervous diseases than females, and that the average preponderance of the stronger over the weaker sex amounts in this respect to nearly 1½ per cent. of the entire mortality from all causes.

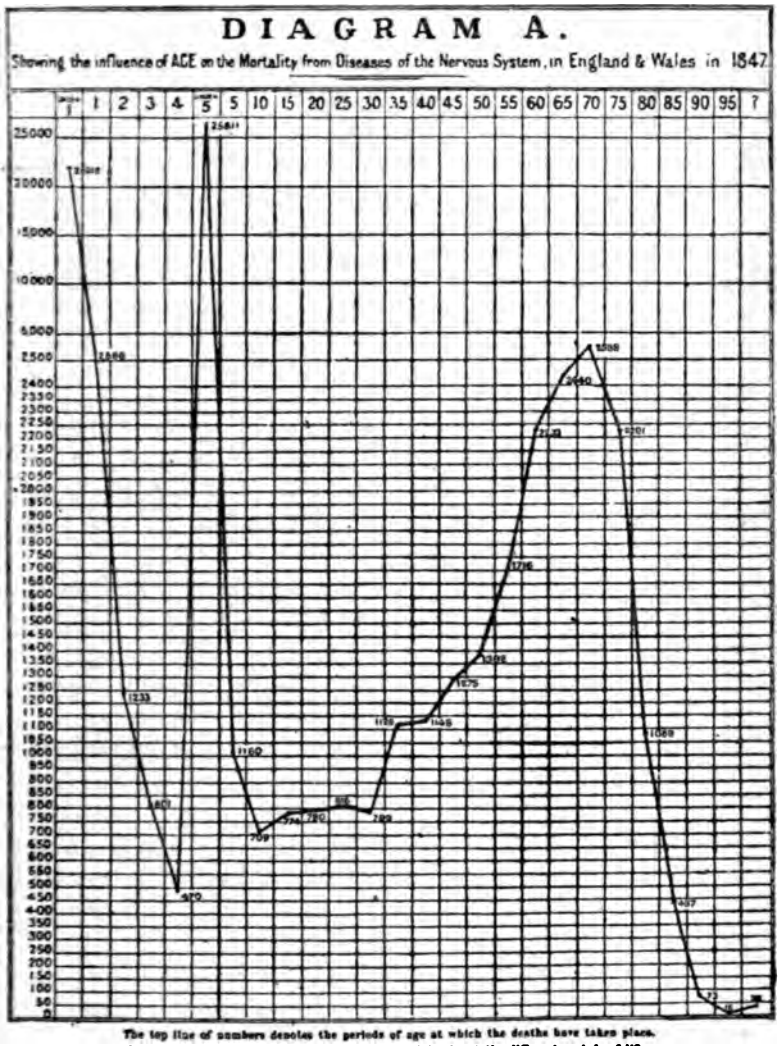
Fifth Proposition.

Age has even a more powerful influence on the production of nervous diseases than sex; for these maladies attain an immense maximum in the first year of life, owing to the great prevalence of infantile convulsions. They are much less frequent in youth and middle age, and attain a second maximum in old age—that is, after seventy, owing to the prevalence of apoplexy and paralysis; but the second maximum amounts to only about the tenth part of the first maximum attained during infant life.

The information given on this point by the Registrar-General applies, as a rule, only to London; and in the earlier volumes is given for twenty-six, and in the later for seventeen different periods of life. For the year 1847, however, the ages of all persons who died in the whole of England have been detailed; and I have thought it best to take the information given for that year as the base for my diagrams, as I consider it more illustrative of this matter than taking only the ages of people who died in London, although for a series of years.

Diagram A shows the influence of age on the mortality from nervous diseases altogether; and we may see at a glance that this influence is far greater than that of sex. The periods of life are stated at the *top*, and the number of deaths which occurred at the *side*. The first five years of life are given each one singly, and then there is an entry comprising all the deaths which have taken place under five years of age. This period is separated from the other periods by a thick stroke. All the other squares contain periods of five years up to ninety-five; after which there is one with a mark of interrogation, containing any in which the age has not been ascertained.

The curve which shows the number of deaths in all these



The top line of numbers denotes the periods of age at which the deaths have taken place.
The side line of numbers indicates the numbers of deaths at the different periods of life.

different periods is, in Diagram A, seen to attain an immense maximum in the first year of life; it descends suddenly after that until four years of age; and the further rise assumed by it only sums up, as it were, the results of the first five years. This curve shows that the number of deaths from nervous diseases during the first lustrum of life is greater than that at all the succeeding ages taken together—viz., in round numbers, 25,000 out of a total of 48,000. From five the curve continues at a low ebb until thirty. At thirty-five a rise is perceptible, which becomes large at sixty, and reaches its maximum at seventy; but this second maximum amounts only to about the tenth part of the first maximum attained in the first period, the numbers being about 2,500 against 25,000. The first maximum is chiefly owing to the mortality from convulsions, and the second maximum to that from apoplexy and paralysis.

A moot point in our subject which still remains unsettled is the *classification of nervous diseases*. The most simple, and in a certain way admirable nomenclature, is that which was adopted in 1838 by the Registrar-General in his statistical reports, and which is as follows:—

1. *Cephalitis*. Under this heading are included inflammation of the brain and spinal cord and their membranes—that is, encephalitis, meningitis, myelitis, and spinal meningitis.

2. *Hydrocephalus*, or dropsy of the brain. This disease, which is now generally called meningitis tuberculosa, only remained in our class until 1842, when it was expunged from it, and transferred to the class of tubercular diseases. This being the more suitable pathological place for the affection, I have not tabulated the cases of hydrocephalus for those years in which it appeared amongst nervous diseases, but deducted them from the sum total of deaths, so as to insure, as far as possible, uniformity in my tables.

3. *Convulsions*, spasms, or fits. This entry is more particularly intended for infantile convulsions or eclampsia, which constitute by far the largest cause of mortality from diseases of the nervous system altogether.

4. *Apoplexy*, the apoplectic fit. Apoplexy being merely a symptom, and not a disease, this entry includes not only cases

of severe cerebral hyperæmia and cerebral hæmorrhage, but also sunstroke, and acute intoxication by opium, alcohol, and other narcotic poisons. On the whole, however, fatal cases of cerebral hæmorrhage must, in the nature of things, form the large majority of cases classed under this heading.

5. *Paralysis, palsy, the paralytic fit, hemiplegia, and paraplegia.* This rubric contains chiefly cases of softening of the brain giving rise to hemiplegia, but also of paralysis owing to cerebral hæmorrhage which has not immediately proved fatal, chronic spinal disease, progressive locomotor ataxy (which generally ends in paralysis), infantile paralysis, and shaking palsy.

6. *Delirium tremens*, or potatorum, was, until the year 1858, included in the class of Nervous Diseases, but was then transferred into Order 3 of Class I. (zymotic diseases), and registered, together with intemperance, under the heading of Alcoholism. I have, however, continued to tabulate the cases of delirium tremens until 1871, in order to gain as extensive a survey as possible of the fatality of this complaint, which I look upon much more as a nervous than as a zymotic disease.

7. *Tetanus*, trismus, locked-jaw.

8. *Chorea*, St. Vitus's dance.

9. *Epilepsy*, the falling sickness, includes in these Reports catalepsy and hysteria; but as hysteria is hardly ever fatal, and catalepsy extremely rare, it appears probable that almost all cases which have been registered were such of true epilepsy.

10. *Insanity*, including monomania, dementia, and idiocy.

11. *Disease*, a general term which is made to include softening without paralysis, abscess, tumour, atrophy of the brain and spinal cord, and diseases of the eyes and ears. This rubric forms a kind of lumber-room into which everything is thrown which cannot be registered under any of the foregoing headings.

The nomenclature of Nervous Diseases which has been adopted by the Royal College of Physicians of London is to my mind unfortunate, as it is neither so simple and practical as the one of the Registrar-General, which I have just mentioned, nor does it fully satisfy scientific requirements. To give only a few instances: we find that tetanus, shaking palsy, and chorea

are put down amongst the *functional* diseases of the nervous system. Again, 'paralysis' is described as a disease of the nerves, apart from diseases of the brain and spinal cord; and in the Annual Report for 1874, where the nomenclature of the College of Physicians has been used in addition to the old classification, this 'disease of the nerves' is stated to have carried off not less than 12,503 persons in that one year! Now if any fact is well established in the pathology of the nervous system, it is this, that paralysis from disease of the peripheral nerves is not only very rare, except in time of war, but also hardly ever fatal; and it is much more probable that no death at all occurred in 1874 from paralysis of the peripheral nerves than that 12,503 should have been caused by it. A statement scarcely less surprising is that during the entire year one person in all England and Wales should have died of tabes dorsalis!

The following is the classification of Diseases of the Nervous System which I am in the habit of using in the registration of the cases which come under my care at the Regent's Park Hospital for Epilepsy and Paralysis:—

I. *General Neuroses.*

1. Nervosity, nevrosismus, general hyperæsthesia and convulsibility.
2. Eclampsia.
3. Epilepsy.
4. Catalepsy.
5. Hysteria.
6. Hypochondriasis.
7. Chorea.
8. Tetanus.
9. Vertigo.
10. Insomnia.
11. Tremor.
12. Athetosis.

II. *Diseases of the Brain and its Membranes.*

1. Hyperæmia.
2. Anæmia.

3. Apoplexy (cerebral hæmorrhage).
4. Thrombosis
5. Embolism of cerebral arteries } softening of the brain.
6. Aphasia.
7. Meningitis.
   a. External pachy-meningitis.
   b. Internal pachy-meningitis (hæmatoma of the dura mater).
   c. Lepto-meningitis.
   d. Tubercular meningitis.
8. Encephalitis.
9. Chronic abscess of the brain.
10. Sclerosis.
11. Hypertrophy.
12. Atrophy.
13. Tubercle.
14. Tumours.
15. Syphilitic affections of the brain.

III. *Diseases of the Spinal Cord and its Membranes.*

1. Hyperæmia.
2. Anæmia.
3. Hæmorrhage into the spinal membranes.
4. Hæmorrhage into the spinal cord.
5. Spinal irritation.
6. Spinal exhaustion.
7. Spinal meningitis.
   a. Pachy-meningitis.
   b. Lepto-meningitis.
8. Myelitis.
   a. Acute.
   b. Chronic.
9. Progressive locomotor ataxy.
10. Hypertrophy.
11. Tubercle.
12. Tumours.
13. Syphilitic disease of the spinal cord.

IV. *Diseases of the Cerebro-spinal Nerves.*

1. *Peripheral paralysis.*
   - a. Paralysis of the third nerve.
   - b. „ of the fourth nerve.
   - c. „ of the minor portion of the fifth nerve.
   - d. „ of the sixth nerve.
   - e. „ of the portio dura. facial palsy.
   - f. „ of the spinal accessory nerve.
   - g. „ of the hypoglossus nerve. glossoplegia.
   - h. „ of the vocal cords. aphonia.
   - i. „ of the cervical and dorsal nerves.
     - α. Paralysis of the serratus anticus muscle.
     - β. „ of the dorsal muscles.
     - γ. „ of the abdominal muscles.
   - j. Paralysis of the phrenic nerve.
   - k. „ of the radial nerve.
   - l. „ of the median nerve.
   - m. „ of the ulnar nerve.
   - n. „ of the lumbar and sacral nerves.

2. *Peripheral spasm.*
   - a. Trismus.
   - b. Blepharo-spasm.
   - c. Unilateral facial spasm.
   - d. Spasm in the hypoglossus nerve.
   - e. Spasm in the spinal accessory nerve.
     - α. clonic torticollis.
     - β. tonic torticollis.
   - f. Spasms of the nerves and muscles of the upper extremity.
   - g. spasms of the respiratory muscles.
     - α. Singultus.
     - β. Tonic spasm of the diaphragm.
   - h. Sternutatio convulsiva, convulsive sneezing.
   - i. Spasm in the lumbar and sacral plexus of nerves.
   - k. Tetany.
   - l. Permanent muscular contractions.

3. *Peripheral anæsthesia.*
   - a. Anosmia, loss of smell.

b. Amblyopia and amaurosis (optic neuritis).
c. Loss of taste.
d. Nervous deafness.
e. Anæsthesia of the fifth nerve.

4. *Peripheral hyperœsthesia.*
   a. Headache.
   b. Spinal neuralgia.
   c. Tic douloureux.
   d. Olfactory hyperæsthesia.
   e. Auditory vertigo, Menière's disease.
   f. Cervico-occipital neuralgia.
   g. Cervico-brachial neuralgia.
   h. Ulnar neuralgia.
   i. Intercostal neuralgia.
   k. Mastodynia.
   l. Neuralgia of the lumbar plexus.
   m. Sciatica.
   n. Neuralgia of the coccygeal plexus.
   o. Neuralgia of the joints.
   p. Visceral neuralgia.
5. *Neuroma.*

V. *Special forms of Paralysis.*

1. General paralysis of the insane (dementia paralytica).[1]
2. Paralysis agitans (shaking palsy).
3. Labio-glosso-pharyngeal paralysis.
4. Hysterical paralysis.
5. Lead palsy.
6. Rheumatic paralysis.
7. Reflex paralysis.
8. Peripheral paralysis from injury to nerves.
9. Infantile paralysis.
10. Paralysis after acute diseases.
11. Diphtheritic paralysis.
12. Scrivener's palsy.
13. Paralysis of the bladder.

[1] Only patients suffering from the first stage of this disease are admitted into the Institution.

VI. *Special forms of Anœsthesia.*

1. Cerebral anæsthesia.
2. Spinal „
3. Hysterical „
4. Toxic „
5. Muscular „

VII. *Affections of the Vaso-motor Nerves.*

1. Megrim, sick headache.
2. Angina pectoris.
3. Graves's or Basedow's disease, exophthalmic goître.
4. Unilateral atrophy of the face.
5. Progressive muscular atrophy.
6. Progressive muscular hypertrophy.

I now proceed to give a rapid survey of the present state of the pathological physiology of the nervous centres, as far as it is of importance for my present purpose.

I. *The Spinal Cord.*

The physiology of the spinal cord is only very partially known at present, and recent investigations into it, instead of simplifying the subject, appear to have rendered it rather more complicated than it was previously.

Division of the entire transverse section of the spinal cord causes complete paralysis and anæsthesia in all parts below the lesion; while section of one lateral half causes paralysis of motion in all parts below the section on the same side, and anæsthesia in all parts below the section on the opposite side of the body. This shows that the motor impulses run down the cord on the same side from which the motor roots emerge, while the sensory impressions run up to the brain in the opposite side of the cord.

Complete paralysis of motion is also caused by dividing the antero-lateral columns and the grey matter in the centre of the cord; while section of the posterior columns and the grey matter in the centre does not lead to this result. From this it has been concluded that the antero-lateral columns are the

motor paths in the cord; this view, however, is controverted by the fact that section of the antero-lateral columns alone only diminishes voluntary motion for a very short time, but does not destroy it, and that this becomes very speedily re-established, if the grey matter in the centre remains uninjured. The function of the anterior columns alone, which were formerly believed to be the true centres of motion, is still absolutely in the dark. They respond freely to stimulation, but conveyance of motor impulses is possible through the grey matter alone, and even through small portions of its transverse section. This substance, while it freely conducts motor impulses, is itself unexcitable to any stimuli of whatever nature, and Schiff has therefore termed it '*kinesodic*.'

With regard to sensation we find that the paths for the senses of touch, pressure, temperature, and tickling are situated in the posterior columns. Section of these destroys the sense of touch in all parts below the lesion, but it does not destroy all sensation, inasmuch as hyperæsthesia, especially to painful impressions, is the result of this operation. The sensation of pain is not conducted through the posterior columns, but through the grey substance; and division of the latter causes analgesia or loss of the sensation of pain, while if the posterior columns at the same time remain uninjured, the sense of touch is preserved. The entire transverse section or any portion of the grey matter may still convey sensations after the antero-lateral as well as the posterior columns have been divided; and as this substance is at the same time not excitable to stimulation, it appears to be not only kinesodic, but also *œsthesodic* (Schiff). Recent experiments of the lumbar cord of rabbits by Woroschiloff have, however, rendered it probable that the lateral columns also play an important part in the conduction of sensations. While, therefore, motor impressions run down the same side and sensory impressions in general run up in the opposite side, more especially of the dorsal and cervical portion of the cord, it is not yet settled whether the paths of all kinds of sensations are crossed, as it would appear that the paths for the sense of touch and the muscular sense run up the same side of the organ.

A singular consequence of section of one lateral half of the

cord, was first observed by Dr. Brown-Séquard, who found that, if guinea-pigs were kept alive after such an operation, they became within about three weeks subject to epileptic attacks, with loss of consciousness. After this condition had once become developed, fits could be produced at any time by irritating a certain area of the skin of the face and neck termed the 'epileptogenic zone.' This zone comprises a region defined by a line proceeding from the eye to the ear, then turning of from the ear to the centre of the lower jaw, branching off from there to the neck, and returning by a semi-circle to the ear. In this zone there is incomplete anæsthesia of the skin, and slight irritation, such as blowing upon it or pulling the hair growing on it, will give rise to epileptic fits. After a time such attacks come on spontaneously, and may occur in rapid succession; but ultimately the epileptic condition and the anæsthesia of the epileptogenic zone is found to vanish. The young of guinea-pigs, born while the condition existed in the parent, inherit the tendency, without having undergone any operation for its production.

The same phenomena have been produced by division of the posterior columns, posterior cornua, lateral columns, the posterior roots of the nerves for the lower extremities, of one or both sciatic and popliteal nerves; also by complete section and by simple puncture of the cord, more especially between the eighth dorsal and second lumbar vertebra. Injury to the medulla oblongata, the pedunculi cerebri, and the corpora quadrigemina, have given rise to the same results; and it appeared that when the cord or the peripheral nerves were divided, the epileptogenic zone was on the side of the lesion, while if the cerebral parts mentioned were injured, it was present on the opposite side.

It cannot therefore be concluded from these facts that the spinal cord has any special relation to the production of epilepsy. Westphal found that guinea-pigs were seized by general convulsions on receiving a light tap on the head. After that the animals continued apparently quite well, but some weeks after the tap had been inflicted, became subject to epilepsy, which lasted from six weeks to six months. The autopsy showed small areas of hæmorrhage in the medulla oblongata and cervical portion of

the cord. Hitzig produced epileptic convulsions by destroying the cortical centre for the anterior extremity in dogs; and it follows from these researches that destruction of the most various portions of the nervous system may give rise to the development of the epileptic condition.

One of the most important functions of the spinal cord is its *reflex action*, which since its discovery by Marshall Hall and Johannes Müller, has been a favourite subject of study for physiologists. This faculty resides in the ganglion cells of the grey substance in the centre of the cord, where sentient impressions are conveyed from the posterior roots to motor paths in the anterior roots, without any simultaneous action of the will. The course of reflex action is probably extremely complicated, and Helmholtz has found that the time required for its occurrence is about twelve times as long as that which is necessary for simple motor conduction.

The intensity of reflex action corresponds directly to the intensity of the stimulus, and inversely to the resistance to passage which is encountered. It may, therefore, be increased by increase of stimulus as well as by diminished resistance to passage. Pflüger has shown that a stimulus of feeble intensity is only conveyed from a sentient fibre to a motive fibre of the same side and at the same level of the cord; while if the intensity be increased, the stimulus is propagated to the corresponding fibres of the opposite side. Further increases cause propagation to the upper, and then to the lower portions of the cord, while ultimately all the muscles of the body may be thrown into reflex contractions.

Reflex action is generally caused by stimulation of the skin; but Erb and Westphal have lately found that it may also be produced from tendons by giving them a light tap with the finger. This is most easily seen on the tendon of the rectus femoris, at the ligamentum patellæ (Westphal's Unterschenkelphænomen), but may also be produced on the tendon Achillis, and that of the triceps muscle of the arm. The muscular contraction which answers to the tap, takes place only in the muscles or set of muscles which are connected with the tendon, and is not owing to mechanical concussion of the muscular substance, as Westphal at first believed, but to real reflex action, the

centre for which is in the lower portion of the spinal cord. The skin does not participate at all in this process. Where reflex excitability is unduly exalted, a similar effect may be produced by giving a tap on the periosteum of certain bones, as also fasciæ and articular ligaments.

Similar reflex actions may be caused from sentient nerves of the muscles, and from the nerves of the viscera, such as the stomach, intestines, bladder, rectum, &c.

Reflex action may be *inhibited* partly by cerebral influence, as we shall see presently when the function of the corpora quadrigemina comes under discussion; and partly through irritation of the sentient nerves of the skin, or of the sensory nerve-trunks, or the sensory nerves of the muscles and viscera; all of which may cause diminution, postponement, and even total abolition of reflex activity.

The spinal cord has likewise great influence upon the *co-ordination of movements*, by which certain muscles or sets of muscles are made to carry out complex actions which have a certain purpose. Co-ordination is composed of two several kinds of action, viz. of automatic or voluntary associations of muscles for a certain end, and of a state of equilibrium between the antagonists. Most movements which occur in daily life are devoid of certainty and precision without co-operation of antagonists; so that one set of muscles produces, and the other regulates and tempers, the movements. Complex muscular movements are learned in early life by daily, nay, hourly practice. Immediately after birth the only co-ordinated movements are those of sucking, swallowing, and breathing; all other movements of this kind are only acquired by constant training. The baby that first begins to walk, may be said to suffer from want of co-ordination of the muscles of the legs (locomotor ataxy), and does not learn to walk or stand without having often fallen down, and thus received many practical lessons of the importance of a judicious association of muscles. In the same manner, speaking, writing, sewing, playing on musical instruments, and other complex movements have to be laboriously acquired, until at last the most intricate muscular actions may be performed instinctively or automatically, without much, if any, effort of volition. Even, however, after

perfection has been attained, the attention of the mind is still requisite, and unless this be given, the most perfect talker, writer, and player will have slips of the tongue and pen, or play wrong notes. We have to assume that the centres for the co-ordination of movements are formed in the embryo, and are capable of development, but that their full faculties are only acquired by years of practice. This appears to diminish the resistance to the passage of the influence of volition to these centres, which from being at first considerable, like that of a country road full of impediments to the driving of a coal waggon, becomes finally as slight as that which is experienced by a well-made brougham on a noiseless London pavement.

The centres of co-ordination are however not actually situated in the spinal cord itself: on the contrary, their seat is in the brain; which is not only shown by the fact that attention is required, even late in life, for carrying out the most complex movements in the most perfect manner, but also by direct physiological experiments, which go far to prove that the seat of these co-ordinating centres is in the cerebellum, the corpora quadrigemina, and the thalamus opticus.

Nevertheless the spinal cord plays an important part in co-ordination, inasmuch as it conducts the impulses for it from the brain to the anterior roots of the spinal nerves. The exact locality of these co-ordinating paths has not yet been ascertained. Pathological facts, more especially such as are observed in progressive locomotor ataxy, render it probable that they are situated in the posterior columns of the cord; while physiological experiments appear to locate them in the middle third of the lateral columns, between the anterior and posterior ones.

The spinal cord also contains centres and paths for the conveyance of nervous impulses to the *viscera*. Those viscera which are under the most immediate influence of this organ, are the bladder, rectum, and the male and female organs of generation.

The influence of the cord upon the movements of the *bladder* has been chiefly investigated by Budge and Goltz. It appears that as urine accumulates in the bladder, the coats of the viscus are stimulated by distension and pressure; this causes reflectory contraction of the detrusor urinæ, the path for it being through

a centre in the lumbar portion of the cord. The desire to pass water is now consciously perceived, and the urine may then either be passed voluntarily by the impulse for the contraction of the detrusor and the relaxation of the sphincter arriving from the brain through the cord in the nerves of the bladder; or the desire may be resisted by voluntary contraction of the sphincter, until the detrusor becomes fatigued or accustomed to pressure, and the desire to empty the bladder passes off. As, however, the urine continues to accumulate, fresh contractions of the detrusor are caused, until the sphincter is relaxed either by the orders of volition, or by the preponderating contraction of the detrusor, and the bladder is emptied. Pressure by the abdominal muscles, either voluntary or reflectory, may considerably promote the evacuation of the viscus.

The centre for the movements of the bladder being situated in the lumbar cord, the viscus may still be emptied even after the dorsal portion of the cord has been divided, as soon as the bladder is sufficiently full or its coat stimulated in any way. Nevertheless the bladder generally appears paralysed in the first few days after the operation, which is owing to shock to the lumbar cord, resulting from the operative proceeding. As soon, however, as the effects of shock have passed off, the function is re-established.

While stimulation of the coats of the bladder itself is the most effective mode of causing the viscus to be emptied, stimulation of the anus and rectum may also conduce to this; and a therapeutical illustration of this physiological fact is, that in obstinate cases of retention of urine, relief may be given by filling the rectum with ice, after all other treatment has failed.

The centre for the bladder in the lumbar portion of the cord is, however, not the only one for it, since Faradisation of the restiform bodies and the pedunculi cerebri likewise cause contraction of the detrusor and evacuation of the viscus. Fibres proceed from the parts mentioned through the anterior columns of the cord to the anterior roots of the third and fourth sacral nerves, and their function may be excited apparently by cerebral influence as well as reflex action, the paths for this being in the posterior roots of the sacral nerves.

In spite of the existence of this cerebral centre, Goltz

inclines to the opinion that volition has no direct influence upon the contraction of the detrusor; and that, if we are able to empty the bladder voluntarily without there being any desire to do so, this is effected partly by relaxation of the sphincter, and partly by powerful pressure of the abdominal muscles upon the coats of the viscus, which in its turn causes reflectory contraction of the detrusor.

The same reflectory centre in the lumbar cord which regulates the movements of the bladder, also serves for those of the *rectum*, as shown by the effects of Faradisation. The fæces on arriving in the rectum cause peristaltic action reflexly; but their evacuation is at first prevented by increased contraction of the sphincter. At the same time the conscious cerebral cortex is informed by the sentient fibres of the rectum that evacuation is approaching; and this latter may then either take place, by the sphincter being voluntarily relaxed, and the reflex stimulus increased through simultaneous voluntary contraction of the abdominal muscles; or it may be prevented for some time by voluntarily increasing the contraction of the sphincter. Ultimately, however, the tone of the latter will be unable to resist the constantly increasing peristaltic motion of the muscular coat of the rectum. As the fæces pass through the anus, reflectory rhythmical contractions of the sphincter ani are produced, by means of which the rectum is again closed up.

Relaxation of the sphincter of the bladder commonly takes place without relaxation of the sphincter ani, while the latter cannot, as a rule, be relaxed without simultaneous relaxation of the former. When, however, the reflex irritation preceding the evacuation of the fæces has reached a high degree, and is at the same time resisted, a simultaneous desire to empty the bladder has likewise to be repressed, unless on relaxing the sphincter of the urethra, that of the anus may follow. Another illustration of these physiological relations is, that spasmodic retention of the urine is best treated with an effective purge; for when the sphincter ani relaxes, the spasm of the urethral sphincter cannot persist. Both sphincters may be paralysed together, without implication of any other part, as is found for instance in certain cases of spina bifida.

The same reflectory centre in the lumbar cord which rules

over the movements of the bladder and rectum, excites the processes of *erection* and *ejaculation*. Stimulation of the erectile nerves which arise from the sacral plexus and proceed to the corpora cavernosa, causes an inhibitory influence upon the ganglionic masses of the bloodvessels of the penis, whereby vascular tone is diminished, and the corpora cavernosa filled with blood to excess. That the erectile nerves are excited by the centre in the lumbar medulla, is shown by the fact that destruction of this centre renders erection impossible, while after the dorsal cord has been divided, erection may still be caused reflexly. There is no direct influence of volition over erection, which cannot be produced at pleasure; but it is generally owing to peripheral irritation in the neighbourhood of the organs, or to stimulation of the imagination. The seat of the sexual desire is in the cortex of the brain, and the reflex centre in the lumbar cord receives its stimulation from there, the path for the latter being through the spinal cord. Faradisation of the upper portion of the cord, the pons, and crura cerebri likewise produce erection. Ejaculation is under the influence of the same lumbar centre, but a more powerful irritation of this latter is required for ejaculation than for erection.

In crushing of the spinal cord from fracture of the vertebræ we generally observe *priapism*, which is paralytic and passive. The erectile tissue of the corpora cavernosa of the penis being unable to contract, blood is pumped into them to excess. There is, however, rarely complete erection, for the organ, although turgid, generally remains flexible. Priapism is greater in crushing of the upper dorsal than of the cervical spine, and is more marked and lasts longer in the young than in the aged.

The reflex lumbar centre likewise regulates the contractions of the *womb* during labour; and parturition may therefore still take place after destruction of the dorsal cord. There are other centres for the uterus in the upper portions of the spinal cord and the medulla oblongata, but none of them are of such essential importance for the induction of uterine contractions as the lumbar centre.

The influence of the spinal cord upon the *digestive organs* has not yet been thoroughly ascertained, but Goltz has rendered it probable that the peristaltic movements of the stomach and

intestines are under the influence of it, inasmuch as after the destruction of the cord powerful peristaltic contractions and diarrhœa take place. The cord also seems to contain inhibitory centres for the œsophagus and stomach.

The *chief vaso-motor centre* is situated in the medulla oblongata, but there are minor centres for the bloodvessels throughout the entire length of the spinal cord. Faradisation of this organ causes constriction of all arteries below the seat of irritation, while destruction of it produces dilatation of the same arteries, likewise below the seat of the lesion. These vaso-motor centres appear to be situated in the anterior cornua of the grey substance of the cord, and the vaso-motor nerves coming from them proceed from the lateral columns through the anterior roots of the spinal nerves to the periphery.

That such centres exist all the way down in the cord is shown by the following phenomena: after the arteries have for some time remained distended from section of the cord, they contract again so as to resume their normal diameter, when the effects of shock, and consequent paralysis of the lower centres, have passed off. Every further section of the cord lower down causes the same phenomena, viz., at first dilatation, and afterwards contraction, of the arteries. Even after destruction of the lumbar cord, the bloodvessels of the hind parts do not remain permanently or completely paralysed; and we are therefore led to the conclusion that, independently of the vaso-motor centres in the medulla oblongata and the spinal cord, there are local ganglionic centres in the vessels themselves, analogous to the motor ganglia of the heart, which still cause rhythmic contractions of that organ, after it has been severed from its centre in the medulla oblongata, as well as from the sympathetic and pneumogastric nerve. After destruction of the higher centres, the lower, and also the peripheral centres, seem to gain in power, through the increased quantity of blood contained in the vessels after such operations.

The cervical portion of the cord also contains a *centre which regulates the production of the body-heat*, and corresponds in its situation to the sympathetic fibres which pass from the medulla oblongata through the spinal cord to the cervical ganglia of the sympathetic nerve. In fracture of the spine and

crushing of the cervical cord, there is, as a rule, greatly increased temperature, owing to, or at least connected with, paralytic dilatation of the bloodvessels. In the earliest stages of such an injury there may be coldness owing to shock, but in a short time the thermometer runs up to 104°, and often shows 110° shortly after death. In exceptional cases the temperature is lowered, so that with the bulb in the urethra or rectum, we only find 93° or 94°. In such cases we must assume that either the effects of shock predominate largely, or that the heat-regulating centre may have escaped the injury. If the patient survives, the temperature is ultimately again lowered in all cases. This is seen eventually after all nerve-lesions, whether motor, sensory, mixed or vasomotor, and is no doubt connected with a reduction of the cellular changes to which the temperature of living tissues is due. If the injury to the spine is confined to one side of it, the increase of temperature is likewise limited to that side; and this is also the case where the brachial plexus is torn across.

The cervical cord is the only organ in the body, mechanical injury of which has the effect of inducing hyper-pyrexia. The centre for the regulation of the body-heat loses its power in lessening combustion also under the influence of certain morbid poisons, such as those of typhoid and rheumatic fever, and of excessive external heat. Such loss of control does indeed appear to be the essence of fever; and as the power of the centre is at the lowest ebb at the time of death, the post-mortem increase of temperature in certain pathological conditions affecting it, is more easily explained with reference to this than could be done in any other manner.

Crushing of the cervical and upper portion of the dorsal cord is likewise followed by contraction of the pupil.

That the movements of the *iris* are partly under the influence of the spinal cord, was already known to Budge, who has described the *cilio-spinal centre* as situated between the seventh cervical and sixth dorsal vertebræ. Faradisation of this region causes the pupil to become dilated, the stimulus being conveyed to the cervical sympathetic nerve, which animates the radiating fibres of the iris (musculus dilatator). After section of the sympathetic nerve the pupil becomes con-

stricted, since the circular fibres of the iris now obtain full power over that membrane. Salkowski has located this centre higher up, in the medulla oblongata, and finds that the motor fibres emanating from it run down in the cervical cord without crossing over, and proceed through the anterior roots of the lower cervical and upper dorsal nerves into the cervical sympathetic, from whence they proceed to the eye. Section of these fibres causes constriction of the pupil, as above.

Trophic disturbances of peripheral parts, after their connection with the spinal cord has been destroyed, are so common that we are obliged to assume the existence of trophic centres in that organ; but nothing definite is known regarding their position and connections. The existence of a special set of trophic nerves, which had been asserted by Samuel, has not been confirmed. It is, however, probable that the trophic centres for the sentient nerves are situated in the spinal ganglia, and that those for the motor nerves and muscles, the joints and bones, lie in the anterior cornua of the grey substance; while the cord itself contains its own centres for its nutrition and preservation.

Respiration and the *heart's action* are more directly under the influence of the medulla oblongata than that of the spinal cord; yet the cord unquestionably participates in regulating these movements. Rokitansky the younger has been led to assume the existence of respiratory centres in the cervical portion of the cord, the function of which is said to become more marked after separation from the medulla oblongata. The paths by which the respiratory impulses travel from their centres to the respiratory muscles, appear to be situated in the lateral columns of the cervical and upper portion of the dorsal cord. Some physiologists are inclined to put the exciting centre of the heart's action into the cervical spine, from where the impulses would travel down to the sympathetic nerve and the heart; but further observations appear necessary for deciding this question.

Accidental crushing of the lumbar and dorsal spine in man, from fracture of the vertebræ, appears to have no influence on the heart's action. Where, however, the cervical spine is injured, the pulsations are diminished, from paralysis of the

sympathetic fibres coursing in that region. The pulse is therefore full and large, and uncommonly slow, as it may sink to thirty-five beats in the minute. This is owing to loss of balance between the several systems of cardiac nerves, and preponderance of the inhibitory, which is represented by the pneumogastric nerve, over the exciting or accelerating nerves, represented by the sympathetic system. It forms a striking contrast to see symptoms of the utmost excitement, distress, and suffering from spinal injury associated in this manner with a slow and steady pulse.

Whether the spinal cord has any direct influence upon the *secretions* remains still unsettled. Eckhard has observed that section of the cervical cord causes an arrest of urinary secretion, and thinks that there is a centre for exciting urinary secretion in the rhomboid fossa. The altered condition of the urine in myelitis and other diseases of the spinal cord was formerly ascribed to a special influence of that organ upon urinary secretion, but is now generally acknowledged to be owing to cystitis from accumulation of urine in the bladder.

Finally we must allude to the law of substitution, or *vicarious interchange of functions*, which Schiff has endeavoured to establish for the several portions of the spinal cord. He has found that after injury to this organ function may return without the anatomical lesion having been repaired; and explains this by assuming that other portions of the cord, which have remained in their normal condition, undertake the duties of the injured portions in their turn. The only exception to this rule is found in the posterior columns, injury of which will permanently destroy the sense of touch, without the possibility of recovery. Whether severe anatomical lesions, such as inflammation, sclerosis, hæmorrhage, etc., are capable of being thoroughly repaired, remains however very doubtful. It is true that excised portions of the cord may be regenerated, in frogs, as shown by Brown-Séquard, Masius, and Vaulair; but such does not appear to be the case in the higher mammalia and man, for Eichhorn and Naunyn could not discover any regeneration of destroyed ganglion cells in dogs. If the lower portion of the dorsal cord was divided, there was complete degeneration of the parts operated upon; afterwards an inter-

mediate substance of cellular tissue, resembling the neuroglia, was formed, in the centre of which there was a cavity; and in the course of many months a scanty number of nerve-fibres were re-formed in this substance. In accordance with these anatomical lesions, partial recovery of motion and sensation took place; but the animals nevertheless perished ultimately in consequence of the injury they had received. Goltz and Freusberg, on the other hand, have never been able to see either regeneration of tissue or recovery of function in dogs which had been treated in this manner; and this latter result would agree with our clinical experience, which shows that recovery from pathological lesions of this sort in man is extremely rare, and that even where it occurs, it is very imperfect.

II. *The Medulla Oblongata.*

This organ, which connects the spinal cord with the great ganglia of the brain, and allows decussation of the motor conductors of the cord in the anterior pyramids, so as to connect each half of the brain with the opposite half of the body, is a centre of reflex co-ordination endowed with the most various and important functions. The principal one of these is to guide and direct the *respiratory movements*. When the entire brain above the medulla oblongata has been removed, respiration will still continue, while if the medulla be destroyed in a warm-blooded animal, this will instantly cease breathing and die. The exact seat of the respiratory centre is in the apex of the fourth ventricle, on the beak of the calamus scriptorius, and has by Flourens been termed the vital knot (*nœud-vital*). Faradisation of this spot causes sudden rigidity of the respiratory muscles of the neck, chest-walls, and diaphragm; and morbid stimulation of it has the same effect. The access of air to the windpipe and the bronchial tubes is prevented, and respiration suddenly arrested, by convulsions proceeding from the medulla oblongata. The brain is consequently all at once deprived of oxygen, and death from asphyxia must ensue unless the convulsion quickly subsides. When this tonic rigidity of the respiratory muscles continues beyond a minute or two, a fatal result is certain; but in many attacks of eclampsia it is followed by clonic convulsions: we

perceive jerking movements of the chestwalls, from alternate rigidity and relaxation of the respiratory muscles, whereby a small quantity of air is allowed to enter the lungs. This at once cuts short the asphyxia, since even a slight supply of oxygen is sufficient to keep up life for a time. The clonic convulsions at first succeed each other rapidly, and then at longer intervals, the contractions becoming less frequent, and the relaxation more prolonged, until at last the rigidity ceases altogether; respiration becomes regular, the attack is over, and the patient's life saved—at least for a time.

The relation of the medulla to respiration explains the curious phenomenon of the cry or scream, which so often ushers in epileptic and other convulsions. This is not, as commonly believed, a sign of pain, but simply a convulsive expiration, owing to sudden irritation of the co-ordinating centre of the respiratory movements.

The medulla oblongata has also an important influence upon the *heart's action*. It does not of itself cause the rhythmical contractions of that organ, which are under the influence of the motor ganglia, situated in the cardiac substance itself. The heart, therefore, may continue to beat rhythmically after all the nerves which connect it with the brain and spinal cord have been divided. The nerves proceeding from the medulla oblongata to the heart have a twofold influence, viz. to accelerate and to restrain its rhythmical action. The restraining or inhibitory influence is exercised by the pneumogastric nerve, section of which causes the pulsations to become so quick that they can no longer be counted; while Faradisation of it, by putting into full force its inhibitory influence, causes the hear to stand still during diastole. On the other hand, the sympathetic fibres which pass from the medulla oblongata down the spinal cord, and reach the heart through the lower cervical and first dorsal ganglia of the sympathetic nerve, accelerate the heart's action. Convulsions proceeding from the inhibitory centre of the heart's action cause an immediate arrest of the cardiac movements, and may thereby produce death by syncope.

The medulla oblongata contains likewise the *vaso-motor* centre, which regulates the innervation of the bloodvessels, and appears to be situated on each side of the median line of the

floor of the fourth ventricle. Section of this centre causes paralysis of the bloodvessels, while Faradisation of it causes their contraction. Convulsion implicating this portion of the medulla causes instantaneous anæmia (or rather ischæmia) of the brain, with the symptoms of vertigo, general convulsions, and apoplectic coma.

The medulla oblongata is further the co-ordinating centre of the movements of *deglutition*, for which it associates the action of the muscles of the lips, tongue, palate, and pharynx. Section of the medulla renders swallowing impossible. Convulsive movements of deglutition are not unfrequently observed together with general convulsions, and then point to irritation of this particular centre. The medulla is likewise the centre for the reflex co-ordination of actions in the sphere of the portio dura, causing reflex *emotional* expression of the face. Facial spasm in its most horrible forms is often combined with convulsions arising from irritation of this portion of the nervous system.

The medulla oblongata is finally the co-ordinating centre of *articulate speech*. It contains the nuclei of the pneumogastric and accessory nerve, that is, the sensory-motor nuclei for respiration and the voice, which are indispensable for speaking; and the motor nuclei of the hypoglossus and portio dura, which organise articulation. The nuclei of these nerves are situated close together, in pairs, at both sides of the raphe; and in consequence of this arrangement, aided by commissural fibres proceeding from one side to the other, a simultaneous contraction of the corresponding muscles of the tongue, lips, and palate may be caused, even when the motor impulse proceeds from one side only.

These anatomical facts are of great importance in the consideration of that form of paralysis which is known as progressive bulbar paralysis, and in degeneration of the grey substance of the spinal cord which has the tendency to ascend to the medulla oblongata. The integrity of pronunciation of letters is bound to the integrity of the motor nuclei in the medulla which have just been mentioned. As in the diseases just alluded to, one ganglion cell after another perishes, pronunciation of one consonant and vowel after another becomes impossible, and the

words indistinct and unintelligible. In such cases there is no affection of the intellect, nor any loss of intelligent language (aphasia), but simply annihilation of speech from impossibility of pronouncing letters. Aphasia is generally associated with agraphia, or loss of the power of intelligent writing; while in progressive bulbar paralysis, the patient is able to write and thereby to communicate his thoughts and sensations, provided the paralysis does not affect the arms and hands likewise.

III. *The Brain.*

Lesions of the *cerebellum* cause disorders of movement resembling those observed in the various degrees of alcoholic intoxication, so that the proper equilibration of the body is interfered with. Faradisation of this organ produces movements of the eyes in different directions, according to the parts of the cerebellum which are acted upon, and also certain movements of the head and limbs, together with contraction of the pupils. These movements are intended to maintain the equilibrium of the body, so that if there be a tendency to displacement, a compensatory action is excited, preventing or neutralising the same. Thus, destruction of the *anterior* part of the middle lobe of the cerebellum causes a tendency to fall forward, while Faradisation or pathological irritation of the same induces such muscular movements as would counteract that tendency. Destruction of the *posterior* part of the middle lobe creates a tendency to fall backwards, and of the lateral lobes to fall sideways, while Faradisation of those parts appears to call into play those muscular actions which are intended to prevent the tendency to fall backwards or sideways. Pathological lesions which destroy the harmony of the different parts concerned in the maintenance of equilibrium, cause the feeling of vertigo. These functions of the cerebellum cannot, however, be quite detached from those of the optic lobes and pons Varolii, and these three parts seem to form a conjoint mechanism incapable of being disjoined without causing a general disorder of function (Ferrier). There appears to be no distinct relation between the cerebellum and the sexual appetite. No doubt priapism has occasionally been observed in hæmorrhage

of the middle lobe of the cerebellum, but such an effect is most likely owing to the pressure of the clot upon the posterior surface of the medulla and the pons.

Injury of the *pons Varolii* causes complete facial paralysis on the same side, and paralysis of the arm and leg on the opposite side. The pons has also an important influence on articulation, as it is found that in disease of it, although intelligent language does not suffer, yet the pronunciation of words is so clumsy that the patients are nearly or quite unintelligible.

Meynert has divided the ganglia of the brain in two great parts, viz., *the ganglia of the tegmentum pedunculi*, which comprise the corpora quadrigemina and the thalamus opticus; and *the ganglia of the pes pedunculi*, which comprise the corpus striatum and the cortex of the brain. The tegmentum and pes are again connected with one another by a special system of commissural fibres.

The functions of the *corpora quadrigemina*, or optic lobes, are of a complex character. When they are destroyed, vision is completely abolished, and the pupils do no longer contract when a light is approached to the eyes. They have also a decided influence on the expression of the emotions, such as fear, terror, pleasure, etc. Faradisation of these bodies causes complex movements of all the muscles of the body, and especially of those which are concerned in the maintenance of the normal attitude, and for purposes of progression. It is also found that the animals in which these parts are faradised, utter peculiar cries or moans; and we may assume with a great degree of probability that the long-continued moaning which is sometimes heard during epileptic attacks, is owing to morbid irritation of the corpora quadrigemina, just as the sharp initial cry of the convulsive seizure appears to arise from irritation of the medulla oblongata.

We have seen that the spinal cord is the centre of reflex action, but it is in this respect subject to a restraining or inhibitory influence on the part of the brain. It is probable that the corpora quadrigemina are the precise part of the brain endowed with this important function; for Setschenow has shown that chemical irritation of these lobes causes a long

interval to elapse between an impression and the reflex movement following it, while otherwise both are almost simultaneous. Powerful irritation of the parts abolishes reflex action altogether; and this is in consonance with the experience of daily life that we are able by strong efforts of the will to prevent, or at least to diminish and postpone, reflex movements. Disease or injury of the inhibitory centre of reflex action increases convulsibility to a very great extent, by the removal of the check physiologically laid upon the reflex function. In decapitated frogs we therefore find reflex excitability enormously increased. The paths for the conduction of reflex inhibition are believed to lie in the anterior columns of the spinal cord.

The *thalamus opticus*, which constitutes the second part of Meynert's ganglia of the tegmentum pedunculi, is insensible to Faradisation. Destruction of it in animals does not cause paralysis or anæsthesia of the skin, but certain motor disturbances which appear to indicate a weakening of the muscular sense. Hæmorrhage into the posterior portion of the thalamus may cause hemiplegia, crossed amblyopia, hemi-anæsthesia, and impaired articulation; these symptoms, however, are not a direct consequence of the lesion of the thalamus, but are owing to the pressure which the clot exercises upon the neighbouring internal capsule of the nucleus lentiformis.

The *internal capsule*, which separates the nucleus from the corpus striatum, contains in its posterior third mixed motor and sensory fibres, which in their further progress towards the hemispheres separate, the motor fibres going forwards, and the sensory going backwards. Charcot and Veyssière have shown that lesions of the anterior portion of the internal capsule cause simple motor hemiplegia, while lesions of its posterior portion cause hemiplegia combined with hemi-anæsthesia. The anterior central hemispheral area, which is purely motor, is called the sphere of the lenticulo-striated arteries; and the posterior central hemispheral area is called the sphere of the lenticulo-optic arteries. Central softening of, or hæmorrhage into, these parts likewise interfere with articulation.

Meynert's '*ganglia of the pes*' consist of the corpus striatum and the cortex of the hemispheres, and appear to determine those movements which are caused by the will and intellect.

Faradisation of the corpus striatum causes unilateral tonic contractions of the muscles of the face, neck, trunk, and limbs, a condition of pleurosthotonus, in which the body is bent to the opposite side with predominance of the flexors over the extensor muscles. It serves as a centre for combined movements, such as running, jumping, etc., which at first require conscious efforts and long-continued training, but ultimately become so easy that they can be performed without much or any attention. The corpus striatum may therefore be looked upon as an auxiliary motor instrument of the will, intended for relieving the cortical centres of voluntary effort and conscious discrimination, of a large portion of the work which would otherwise have to be done by them.

Lesions of the left corpus striatum interfere more with articulate speech than lesions of the right. They may destroy articulation entirely, or cause such difficulty in speaking as to render what is said unintelligible. Loss of articulate speech is therefore generally combined with right hemiplegia; where it co-exists with left hemiplegia, it is generally only temporary and owing to shock. The corpus striatum is the uppermost limit where lesions of the brain cause simply impaired or impossible *articulation*. Persons who have suffered from hemiplegia and loss of speech from hæmorrhage into this central portion, may still write and express their thoughts intelligently with the non-paralysed hand, provided the cortex of the brain is in a state of integrity, while real aphasia, or loss of intelligent language, is always owing to lesions of the cortical substance of the brain.

The highest development of the brain is produced in the *cortical substance of the hemispheres*. The convolutions of the cerebral hemispheres which surround the corpora striata are the centres of conscious motor activity, and as such in intimate connection with all inferior centres.

It was formerly believed that the cerebral hemispheres were insensible to any form of stimulation applied to them, whether mechanical, chemical, thermic, or electrical; and Flourens, who was the pioneer in the physiological investigation of the functions of the brain, laid it down as a general principle that all portions of the hemispheres were capable of fulfilling the same functions,

and might supplement each other in case one or several of them were destroyed. This *law of substitution or vicarious interchange of functions*, which was for a long time accepted by physiologists and physicians, may now however be looked upon as repealed by the efforts of numerous observers, who, although they differ somewhat in detail, agree in the main as to the fact *that certain cerebral functions are bound to certain cerebral areas*. The only physiologist of note who is still opposed to this principle is Dr. Brown-Séquard; and we cannot help indulging a hope that he may, as evidence accumulates more and more, likewise become converted to the true doctrine.

The Localisation of Cerebral Faculties

is at the present time the favourite study of the most advanced observers of all countries, and can only be briefly touched upon in this place; more especially as our knowledge of it is still in its infancy, and present conclusions and theories will unquestionably have to undergo extensive modifications before they may be looked upon as settled.

It is the merit of Fritsch and Hitzig[1] to have shown by physiological experiments that there are true motor centres in the cortex of the brain; that motor and sensorial centres are differently located; that there are definite areas ruling the movements of the front and hind legs, the jaw and the tongue in animals, and that supplementary or vicarious action is only possible within these areas, but not outside them. This they were able to prove by applying electricity to different portions of the cerebral convolutions of living animals. Their experiments made it evident that grey matter could be directly stimulated, and that, according to the locality where the electrodes were applied, movements of individual sets of muscles took place, while areas in close contact with each other reacted in a totally different manner.

Soon after the investigations of the German observers had been made known, Dr. Ferrier[2] commenced a series of experi-

[1] Fritsch and Hitzig in Reichert and Du Bois-Reymond's Archiv., 1870; and especially, Hitzig, 'Untersuchungen über das Gehirn.' Berlin, 1874.

[2] 'The functions of the Brain.' London, 1876.

ments on the same subject, the results of which have been recently embodied in a most able and suggestive volume, and which have greatly advanced our knowledge of the functions of the brain. The results obtained by Hitzig and Ferrier are found to harmonise in many instances with the clinical observations of Dr. H. Jackson, who has for years past endeavoured to trace localised unilateral convulsive movements to morbid irritation of certain portions of the cortex of the brain, producing what he calls 'discharging lesions.'

The objection which was at first raised to Hitzig's and Ferrier's experiments, viz., that the movements observed were not owing to the stimulation of the cortex, but to the diffusion of the electric current to the true motor centres lower down, has been thoroughly disproved. It is now well established that a current of very low intensity, if applied at the proper point, causes certain movements, while a more powerful one, applied at the wrong point, does not produce any such movements. Moreover, if the centre be destroyed, and a current applied a few days afterwards to the raw surface, no effect is produced, although the transmission of the current is promoted rather than impeded by the injury.

Nevertheless no complete unanimity has as yet been brought about amongst those who are best able to judge of these conditions. Thus Schiff, Eckhard, and Hermann deny that the cortex is electrically excitable, and contend that the effect observed is in reality owing to stimulation of the medullary fibres proceeding from the cineritious substance to the central ganglia; and Eckhard has traced one of these excitable medullary fibres for the front leg down to the corpus striatum.

Messrs. Lussana and Lemoigne[1] likewise deny that the cortical centres of Hitzig and Ferrier are motor centres in the ordinary sense of the word. They point to the fact that mechanical stimulation does not excite them, and that Faradisation and galvanisation are generally ineffectual when the animal is anæsthetised, or immediately after death. In all these points the cortical centres differ very much from the spinal cord, the medulla oblongata, and other centres of motion.

[1] 'Archives de Physiologie.' Paris, 1877.

In very young animals the existence of these centres cannot be shown, although reflex action is highly developed in them; and in some adult animals it is likewise impossible to make the cortical centres respond to electrical stimulation. The same observers also point to the fact, which has already been dwelt upon by Goltz, that the motor paralysis which is observed after destruction of these centres, is not complete nor persistent, while after destruction of the lower motor centres there is complete and permanent paralysis. They have therefore arrived at the conclusion that the centres in the cortex are the organs of volition and instinctive faculties; and that if they are destroyed, the movements may still be executed, although without an intelligent purpose.

It appears to us that this question cannot be entirely decided by electrical investigations alone, but that histology, comparative anatomy, and pathology will have to furnish contributions towards the elucidation of it before we can expect to arrive at the truth. Unfortunately, very little has as yet been done as far as the two first-named auxiliary sciences are concerned. We must, however, give a due meed of praise to the researches of Betz of Kiew,[1] who has shown that the surface of the hemispheres is, by the fissure of Rolando, divided into two halves, in the anterior one of which, which corresponds to Hitzig's centres, the microscope reveals colossal pyramidal cells, while in the posterior one, which is not excitable, layers of nuclei predominate. Betz has found that his 'giant-pyramids,' which are found in Hitzig's excitable zone, occur nowhere else; and what is even more important, that they only become properly developed after birth. We have in this distribution of nervous elements, therefore, an analogy with the anterior motor and the posterior sensitive sphere in the grey centre of the spinal cord. A valuable contribution to the same subject has been made by Dr. Herbert C. Major,[2] who has investigated the structure of the island of Reil in apes, and has found the pyramidal cells in it much smaller than in the human insula.

[1] 'Anatomischer Nachweis zweier Gehirncentren.' *Centralblatt.* 1874, p. 578.

[2] 'The Structure of the Island of Reil in Apes.' The *Lancet*, July 14, 1877.

Von Gudden[1] has discovered that in the inexhaustible squirrel, the frontal or motor portion of the cortical substance is much more developed than in the tranquil, lazy rabbit; showing that motor impulses vary according to the development of the anatomical organs provided for them.

A most important fact first brought forward by Hitzig is that, after the electrical stimulation of the grey motor centres has ceased, the movements which were produced under the immediate influence of the galvanic current have a tendency to reproduce themselves either partly or entirely, without further stimulation, but as an after-effect of the same; and that such spontaneous convulsive movements may ultimately develope into epileptiform convulsions of the entire body. This is an important physiological foundation for what has been called 'cortical epilepsy,' and finds its analogy in the convulsive seizures which are observed in patients as a consequence of circumscribed irritation of the cortex by inflammation, tumours, and parasites. Such convulsions are at first partial, and become gradually transformed into true epileptic fits with loss of consciousness.

Important crucial evidence as to cerebral localisation is furnished by circumscribed destruction of motor centres, which appears to cause either temporary or permanent paralysis of those sets of muscles which are ruled by the cortical centres. The principal methods for this investigation are those of Nothnagel, who cauterises small portions of the cineritious substance with chromic acid; of Goltz, who washes cerebral matter out from an opening made by a trephine, by means of a stream of water; and of Ferrier, who destroys brain matter with the actual cautery.

The time which has elapsed since these investigations were undertaken is not sufficient to enable us to judge positively about their results. Yet there is no doubt that in some cases the loss of power is merely temporary; and some difficulty seems to arise as to a satisfactory explanation of this fact.

Does Flourens's law of supplementary or vicarious action find an application in this instance? If function reappears after the destruction of the corresponding centre, we may assume

[1] Kussmaul, 'Die Störungen der Sprache.' Leipzig, 1877.

that the destroyed portion of the cortex is supplemented, either in the same or the opposite hemisphere, by another portion which had already been trained previous to, or is only being trained after, the reception of the injury. If the destruction be small, we may assume vicarious action in the same hemisphere; but if it be extensive, it would be necessary to look to the corresponding centre in the opposite hemisphere as a substitute.

Dr. Broadbent[1] has proposed a theory of bilateral action of the hemispheres, which at first sight would seem applicable to this case. According to him, in hemiplegia from corpus striatum haemorrhage, only those parts are really found devoid of motor power which have the faculty of acting singly, and independently of the corresponding parts of the opposite side. These parts are the arms, which are habitually engaged in totally different motions, and the legs, one of which in walking supports the body, while the other is thrown forward. On the other hand, the muscles which escape the paralysis are those which act only bilaterally, or in concert with the corresponding muscles of the opposite side. Thus it is impossible to expand one side of the chest or abdomen, or to move one eye, without the other.

The anatomical explanation of these phenomena is, according to Dr. Broadbent, as follows: When muscles habitually act together, the nuclei of their nerves are usually connected by commissures, and form, as it were, a single nucleus. This latter receives a set of fibres from each corpus striatum, and is usually called into action by both, but will be capable of being excited by either singly, and that more or less completely, according as the commissural connection between the two halves is more or less perfect. If, therefore, the centre of volitional action is destroyed on one side, the other will transmit an impulse to the common centre, and this will be communicated to the nerves of both sides; equally, if the fusion of the two nuclei is complete, and in this case there will be no paralysis at all; and more or less imperfectly to the nerve of the affected side, where the transverse communication is not so perfect, in which case there will be a slight degree of paralysis, or rather impaired power.

Thus, for instance, the nuclei of the two third cerebral

[1] 'British and Foreign Medico-Chirurgical Review,' April, 1866, p. 468.

nerves are so intimately connected together in the floor of the fourth ventricle, that they may be considered as one single centre; and therefore, if one corpus striatum be left uninjured, the centre will be called into action on both sides equally, so that the muscles of the eye of the paralysed side, which are animated by the third nerve, act as if nothing had happened. On the other hand, the communication between the nuclei of the portio dura is far less perfect, so that the facial nerve on the paralysed side receives a less energetic order through the partial communication between itself and its fellow on the opposite side—the result being that the order sluggishly given is sluggishly obeyed. Again, taking the individual muscles animated by the portio dura and fifth nerve separately, it is found that those which have rather more power to act independently, viz., the straight muscles, such as the levator anguli oris, the zygomatics, the buccinator, are more paralysed than those which act bilaterally, such as the orbicularis oris and palpebrarum. In the same way the muscles of mastication escape almost entirely, as they always act together on both sides.

The abdominal muscles can hardly, if at all, be used unilaterally; but if the body is inclined to one side or the other, the rectus abdominis may act without its fellow, and in similar movements impaired power may be shown in hemiplegia.

Although this doctrine is well suited to explain the phenomena observed in corpus striatum hæmorrhage, it appears difficult or impossible to apply it to the case now under consideration; for it would then be necessary that, if an injury of the motor centre on one side had been followed, first by paralysis, and afterwards by re-establishment of function, bilateral loss of function should ensue, if the corresponding centre in the opposite hemisphere were likewise destroyed. Such, however, is not the case. Goltz[1] has shown that dogs who had first suffered from paralysis of certain sets of muscles of the right side, by having the motor centre in the left hemisphere injured, would, after having recovered from the same, show the same symptoms of paralysis in the left side, after a similar injury to the right hemisphere, but no complete paralysis.

[1] 'Archiv. für Physiologie,' 1876, vol. iii. p. 1.

The following explanation appears to us better suited to meet the facts of the case. Let us suppose the stimulation, like a locomotive engine, to be able to travel by several lines of rails, the most direct one of which is used as a general rule. In case, however, this line should become blocked by accident or obstruction, a more circuitous route by supplementary lines of rails would be chosen for the despatch of the trains, and which, after some more or less ineffectual trial trips, might gradually be worked as easily as the straight line was in the commencement. The circuitous route would in this case be constituted by anastomoses of fibres and commissures.

After partial destruction of the cortex, whether of Hitzig's excitable zone or of the posterior portion of it, certain disturbances of sensibility are observed, which correspond to the symptoms of paralysis that have been mentioned. There is more or less complete abolition of the sense of touch and of pain, together with disturbed vision; and the degree of anæsthesia corresponds to the extent of the injury inflicted. These alterations of sensibility appear permanent in the skin, although they diminish in intensity, and can at a later period be only demonstrated by the finer tests which are usually employed in such investigations. With regard to vision, it is found that there is at first complete blindness at the side opposite to the lesion, but that, as time goes on, the sight is gradually re-established, and only some very singular optic phenomena remain, which are as follows: If such animals are limited to the use of the suffering eye by destruction of the healthy one, they appear to have lost long sight. They may still get out of the way of impediments which are placed on their route, but they proceed onwards without recognising that they have come to the end of the supporting plane; they do not recognise food except by smell, and are no longer frightened by appearances which would otherwise terrify them. The intellect of the animals remains normal all the time. Goltz explains these phenomena by assuming loss of the sensation of colour and locality, while Lussana and Lemoigne, who observed the same appearances in pigeons, think that they are owing to the interruption of the connection between the right eye and the right hemisphere, after the left eye and hemisphere are removed.

The right eye, however, still remains connected with the uninjured mesocephale and cerebellum, so that the motor centres behind the hemisphere continue to act, and to receive impressions from the uninjured eye.

Animals in which small portions of the cortex have been destroyed, suffer from temporary ataxy; if the loss be larger, there is crossed paralysis, which is followed by motor debility, ataxy, and the so-called manège or circus movements. Ultimately there remains a partial or complete loss of the faculty of making those movements for which man uses the hand; as, for instance, 'shaking paws,' cleaning the head, and taking hold of bones. The automatic or mechanical movements, like running, are re-established, but movements learnt by exertion of the intellect are lost or impaired.

Faradisation of the *angular gyrus* produces apparently visual sensations in animals, while its destruction or ablation causes blindness in the opposite eye. The *superior temporo-sphenoidal convolution*, on the other hand, seems to be the auditory centre. Destruction of the same causes deafness, while Faradisation of it makes the animals prick the opposite ear, open the eyes, and turn the head and eyes to the opposite side, which gives them an attitude of listening. The *hippocampal region* is, according to Dr. Ferrier, the centre for tactile sensation, who finds the centres for smell and taste in the *subiculum cornu ammonis*. This part is found particularly developed in animals with a keen smell, such as dogs, cats, and rabbits.

One of the most important cortical functions is that of *intelligent language*; and this has been not only experimentally but also pathologically more thoroughly studied than the preceding subjects. It is now well understood that the infra-cortical areas only serve for the formation and connection of letters in such strength, quickness, and succession as is intended by the cortex. The perception of spoken words as a simple act of hearing, and the perception of written words as a simple act of seeing, is accomplished by infra-cortical areas; but the intelligent appreciation of their connection with thoughts and ideas is a cortical function. This faculty appears to reside not simply in Broca's region, or the third left frontal convolution,

but in a sphere which comprises the island of Reil, with the adjacent frontal, parietal, and temporal region. Within this area, however, the third left frontal convolution is of particular importance, and destructive lesions of it will cause aphasia or loss of intelligent language.

In connection with this it is interesting to notice that Hitzig found the motor centre for the muscles of the mouth, tongue, and jaw in the lowest portion of the anterior central convolution; and that by galvanising this portion, not only uni-lateral crossed, but also bi-lateral movements of the mouth were obtained. From this it would appear that stimulation of one hemisphere is sufficient for producing those movements of the mouth and tongue which are required for speaking; and thus a complete harmony is brought about between physiological data and pathological facts, showing that lesions of one hemisphere are sufficient to cause complete loss of language. Dr. Ferrier localises the centre for the movements of the mouth and tongue a little in front of Hitzig's centre, and in a situation more closely corresponding to Broca's region.

The *occipital lobes* of the brain, which, as Betz has shown, differ in histological structure from that of Hitzig's excitable zone, have also a different supply of bloodvessels, and different functions. The anterior and parietal portions of the hemispheres are nourished by branches of the internal carotid arteries, while the posterior lobes are fed by the vertebral arteries and their branches, viz. the posterior cerebral arteries from the basilar. The vaso-motor supply of these parts is also different; for while the internal carotid artery is supplied by ascending branches of the superior cervical ganglion, the vertebral artery receives its nervous influence from the inferior cervical ganglion, which takes up ascending branches of the thoracic and abdominal ganglia, and is thereby in intimate connection with the liver and other viscera. The posterior lobes do not respond to Faradisation or galvanisation, and their destruction does not seem to interfere immediately with motion, sensation, or any other functions of the life of relation. There is, however, loss of appetite, and the view which would attribute to them systemic or visceral sensation, seems to be in accordance with their vaso-motor connection as well as with the symptoms observed in

anæmia or disease of these lobes, and which are chiefly those of melancholia.

The further elucidation of the cortical functions must be reserved for future investigations. The difficulties of this subject are enormous; for even the ordinary anatomy of the cortex is of very recent date,[1] and the veil which has hung so long over its histological and physiological properties is only just beginning to be lifted to a slight extent. With regard to many so-called proofs taken from pathological cases, we cannot close our eyes to the fact that a very large number of them, more especially at the earlier periods of investigation, have been most insufficiently described. A curious illustration of this is found in a circumstance which occurred to Broca. At the time when there was great excitement in Paris about the localisation of the lesions producing aphasia, Duchenne happened to tell him that Trousseau had just had a case of aphasia at the Hôtel-Dieu, in which the third left frontal convolution had been found uninjured. Broca at once went to that hospital in order to verify the observation, and found on examining the specimen, that Trousseau had overlooked a softening in that very convolution which existed together with softening of the parietal convolution and the island of Reil. Now if a great master like Trousseau failed to notice an important lesion, and just at a time when this question particularly engrossed the attention of the profession, what can be thought of the observations of many of the *dii minorum gentium*? It is this consideration which vitiates completely the line of reasoning taken up by Dr. Brown-Séquard in this matter, who relies principally upon exceptional and probably badly-described cases, some of which occurred thirty or forty years ago. It is therefore indispensable, in judging of these conditions, to act according to Morgagni's precept, *that cases should not be counted, but weighed.*

An attempt has been recently made to utilise the present views of the localisation of the cerebral faculties for the treatment, by the trephine, of injuries to the skull and brain, which are accompanied by certain forms of paralysis and spasm, known

[1] Eckers, 'Convolutions of the Human Brain,' translated by Mr. Galton, London, 1873, should be in the hands of all who take an interest in this subject.

to arise from disease or injury of cortical centres. M. Lucas Championnière and others have directed the attention of the Paris Academy of Medicine to this matter, and laid down certain rules for hitting 'the line of Rolando' with the crown of the trephine. Such proposals and operations appear to be dictated more by enthusiasm for physiological and pathological discoveries than by sound surgical judgment. Professor Gosselin, in a report on this proposal, has clearly shown that any views which we may entertain concerning the localisation of cortical areas cannot alter the general principles which must guide surgeons in their decision on the advisability of using the trephine. In cases of fracture of the skull, with depression, surgeons have not to look for the line of Rolando, but judge, by the condition of the wound and the fracture, whether the trephine is to be used or not. Where patients suffer from symptoms of lesion of the cortical centres after a blow to the head, but have no fracture of the skull, the use of the trephine appears contra-indicated, not only because a cure is possible without an operation, but also because trephining under such circumstances may lead to meningitis, encephalitis, and septicæmia, and would most probably be utterly useless for the purpose it was intended to serve.

I shall now proceed to consider the mode of production of the several diseases of the nervous system taken separately, and shall do so in the order in which I have found them numerically important. While following the classification of the Registrar-General as far as headings are concerned, I shall analyse these pathologically as I proceed, so as to bring them in consonance with the teachings of modern science.

CHAPTER II.

CONVULSIONS.

CONVULSIONS or eclampsia constitute by far the most frequent and fatal disease of the nervous system; for during the thirty years over which my researches extend, more than three quarters of a million of deaths were owing to them in England and Wales. It appears from my tables that nearly one half of the entire number of deaths from all nervous diseases were due to their influence, the total percentage for the thirty years which have been investigated amounting to 47·84 per cent.

The following table shows the exact number of deaths from convulsions which have been registered during six periods of five years each, as well as the percentage of mortality, 1st of nervous diseases, and 2nd of all diseases:—

Periods of five years	Deaths from Convulsions	Percentage of Nervous Diseases	Percentage of all Diseases
1838–42	127,276	60·97	7·35
1843–46	Not registered	—	—
1847–51	117,991	49·26	5·84
1852–56	122,796	47·61	5·91
1857–61	126,602	46·72	5·84
1862–66	131,829	45·04	5·52
1867–71	130,027	42·19	5·15
Total of thirty years .	**756,521**	**47·84**	**5·78**

A glance at this table shows that there has been an uninterrupted fall in the mortality from convulsions ever since registration was commenced. An absolute maximum of over sixty per cent. was attained in the first period; in the second

there was a fall of more than eleven per cent., and this is so great as to suggest the suspicion that registration was not very accurately carried out during the first lustrum. Nevertheless no doubt can exist that the mortality from this disease is really diminishing; and its diminution is so decided that it has allowed a rise to take place in the mortality from other nervous diseases, such as paralysis and insanity, without causing much disturbance in the total mortality from all nervous diseases taken together. The extent of diminution is seen in the following table:—

Fall of mortality from convulsions between 1838 *and* 1871.

From 1st to 2nd period	. .	11·71 per cent.
„ 2nd to 3rd „	. .	1·65 „
„ 3rd to 4th „	. .	·89 „
„ 4th to 5th „	. .	1·68 „
„ 5th to 6th „	. .	2·85 „
Entire fall from 1st to 6th period		18·78 „

This table shows that the fall of the mortality from convulsions has, with the only exception of that from the first to the second period, which is doubtful, increased of late; and as during that period great progress in the treatment of this disease has been made, we are probably justified in ascribing this fall to an advance in therapeutics.

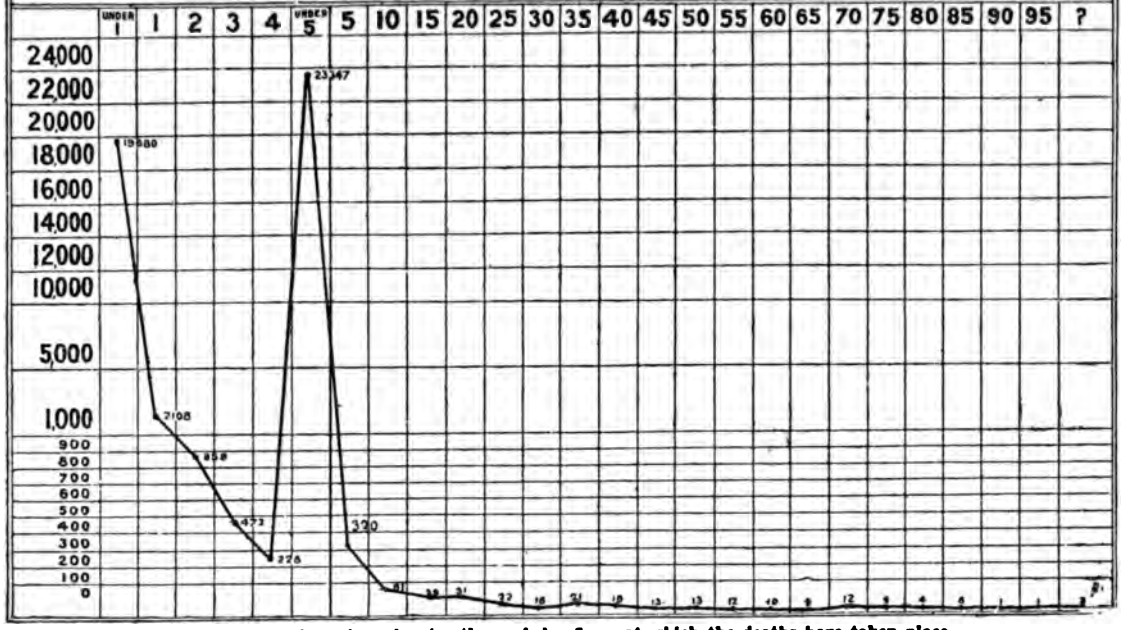
I now proceed to consider the influence of *age* and *sex* upon the production of convulsions.

That infants are more liable to them than adults or the aged has been acknowledged by all previous observers; but up to the present time it has not been suspected that the influence of age upon this disease is as enormous as I have shown it to be. This point is illustrated in a most striking manner by the annexed diagram, in which the curve commences at the top, showing

19,680	deaths	in	the	first	year	of life;
2,108	„	„	second	„	„	;
859	„	„	third	„	„	;
472	„	„	fourth	„	„	; and only
228	„	„	fifth	„	„	.

DIAGRAM B,

Showing the influence of AGE on the Mortality from CONVULSIONS in England and Wales in 1847.



The top line of numbers denotes the periods of age at which the deaths have taken place.
The side line of numbers indicates the numbers of deaths at the different periods of life.

The first period of five years shows altogether a mortality of 23,347, and convulsions are thus seen to be, without exception, the most fruitful source of infant mortality. No other disease equals it in severity, for even 'atrophy and debility,' which comprises the ultimate issue of a number of different diseases, is less fatal than convulsions.

The following is a synopsis of the deaths which the most fatal diseases of infantile life from birth to the fifth year of age caused in 1874 :—

Convulsions	26,534
Atrophy and debility . . .	26,514
Bronchitis	21,147
Scarlet fever	16,712
Pneumonia	13,638
Measles	11,318
Whooping-cough	10,012
Hydrocephalus	5,991
Teething	4,316
Croup	4,220
Diphtheria	1,713
Small-pox	543

The entire infantile mortality (under five years) having been 213,799 in the year 1874, we find that convulsions caused 12·42 per cent. of the entire mortality during that period of life.

After the fifth year the curve in the diagram descends with unparalleled precipitation, never to rise again. The numbers for the different periods of life are as follows :—

Under 5 years of age . . .	23,347 deaths
From 5 to 9 „ . . .	320 „
„ 10 „ 14 „ . . .	61 „
„ 15 „ 19 „ . . .	29 „
„ 20 „ 24 „ . . .	31 „
„ 25 „ 29 „ . . .	22 „
„ 30 „ 34 „ . . .	16 „
„ 35 „ 39 „ . . .	21 „
„ 40 „ 44 „ . .	18 „

From	45	to	49	years	of	age.	13 deaths
„	50	„	54	„			13 „
„	55	„	59	„			12 „
„	60	„	64	„			10 „
„	65	„	69	„			9 „
„	70	„	74	„			12 „
„	75	„	79	„			9 „
„	80	„	84	„			4 „
„	85	„	89	„			6 „
„	90	„	94	„			1 „
„	95			„			1 „

The disease therefore ceases to be important after ten years of age.

So much for the influence of age in the production of convulsions. With regard to the *influence of sex*, opinions differ considerably amongst the ablest physicians, some considering that sex has no influence at all, while others assert that the male or female sex are more predisposed to this disease. This question is, however, absolutely set at rest, at least as far as this country is concerned, by the following table, which shows at a glance the influence of sex on the mortality from this disease in England and Wales. Convulsions are seen to be *much more fatal to boys than to girls*, the mean percentage being 25 for the former, and only 20 for the latter.

Influence of Sex on Convulsions.

Periods of five years	Males	Percentage	Females	Percentage
1847–51	66,280	27·72	51,711	21·59
1852–56	69,070	26·80	53,726	20·74
1857–61	71,132	26·17	55,470	20·40
1862–66	73,966	25·25	57,863	19·76
1867–71	72,784	23·58	57,223	18·55
Mean percentage for 25 years . . .		**25·77**		**20·28**

This table shows throughout the entire period over which

the investigation extends, a decided excess of male over female mortality. Although more boys are born than girls (in the proportion of 104 to 100), yet there is an excess of females over males living. In 1874, for instance, in round numbers, 435,000 boys, and 420,000 girls were born; but the male death-rate was 23·6 per 1,000, and the female 21·0 per 1,000, and the population was estimated in round numbers at 11,512,000 males, and 12,135,000 females, showing an excess of 623,000 females. This excess of females over males is partly owing to the greater fatality of convulsions in boys as compared with girls; other important factors are diseases of the respiratory organs, and deaths from accidents or negligence, which are likewise much more numerous in males than in females. The following numbers show the excess of male over female mortality from convulsions:—

1847–51 . . .	6·13
1852–56 . . .	6·06
1857–61 . . .	5·77
1862–66 . . .	5·49
1867–71 . . .	5·03

The difference between the first and last lustrum amounts to 1·10; and as the excess in percentages has become uninterruptedly smaller, we are allowed to draw the conclusion that *the influence of sex on the mortality from convulsions, although still considerable, is in process of being diminished.*

The term 'convulsions' being an exceedingly indefinite one, I now proceed to show to what class of diseases the term used by the Registrar-General should be applied.

Convulsion means a morbid excess of muscular motion, taking place independently of the will, in the striped or voluntary muscles which are animated by the cerebro-spinal nerves; while by the term *spasm* we understand a morbid excess of motion in the unstriped contractile fibres, which are animated by ganglionic or sympathetic nerves. But not all convulsions occurring in the striped or voluntary muscles can be comprehended in this section; for we have to exclude from them a number of convulsive diseases with special features of their own,

that distinguish them from eclampsia. These diseases are epilepsy, hysteria, tetanus, chorea and hydrophobia.

All convulsions are of *nervous origin*; for although the principal symptoms are shown in the sphere of the striped muscles, which are thrown into a more violent, prolonged, and frequent action than is seen in health, when acting under the influence of the will, yet the inherent property of the muscles to contract, which is known as Hallerian irritability, is of no influence whatever in the production of convulsions. The muscles are nothing but passive instruments, which are put into morbid play through undue excitability of the nervous system that animates them. It is true that inherent muscular convulsions may be caused artificially by the influence of the poison of veratria, which will unduly excite the action of the muscles without the intervention of the nervous system; but this is a form of convulsion which does not occur in practice, and has only been discovered by physiological experiments.

Convulsions in general may be most conveniently classified as *direct*, *reflex*, and *central*. Direct convulsions are those which are owing to increased excitability of a motor or efferent nerve; reflex convulsions are those produced by an irritation of sentient or afferent nerves which is transmitted to the spinal cord; and central convulsions those which are caused by irritation of the cerebro-spinal motor centres themselves.

Direct or local convulsions are owing to an excited state of a motor or efferent nerve at some point of its course between the brain and spinal cord on the one hand, and of the periphery of the body on the other hand. Such convulsions are localised in the muscles which are animated by the nerves in question, and do not extend beyond their sphere unless other portions of the nervous system should suffer at the same time. An instance of this kind of convulsion is convulsive tic of the face, as produced by direct irritation of the facial nerve through rheumatic effusions, or disease of the osseous canals through which the nerve is passing in its way from the base of the brain to the muscles of the face. Convulsions of this kind, although sufficiently troublesome to the patient affected by them, are never fatal, and are therefore not included in the present class.

Reflex convulsions are produced by irritation of the sentient or afferent nerves at some point of their course being transmitted to the spinal cord. Ordinary impressions of this kind will produce physiological reflex movements, such as coughing, yawning, sneezing, laughing, etc.; but if the impression is unduly powerful, convulsion may be the result. Structural alterations of the sentient nerves, such as inflammation; caries of the teeth; or foreign bodies and tumours pressing upon the tissue of the nerves; injury, either accidental or from surgical operations; excessive heat or cold, may, especially when acting on the terminations of the sentient nerves, transmit to the spinal cord an exaggerated impression, which may be followed by equally exaggerated muscular movements.

In health a moderate stimulation will excite reflex action only on that side to which the stimulus is applied. Thus, gentle tickling of the sole of a foot will cause withdrawal of that leg; but if the irritation is more intense it is transmitted to the opposite side of the cord, and the muscular contractions will ultimately occur in all four extremities of the body. We have seen in the first chapter that this transmission takes place through the ganglionic cells of the grey matter of the spinal cord. The pathological phenomena resemble in this respect the physiological ones very closely. Exaggerated muscular movements produced by unduly powerful stimulation are at first generally localised in the muscles of that region from where the over-stimulation proceeds. Where, however, the over-stimulation continues, or where the centre itself is habitually unduly excitable, the reflex convulsions may gradually spread from the local seat of the injury to distant parts, affecting first the corresponding side, but ultimately the whole of the spinal cord, and giving rise to general convulsions of the extremities. When such a state of general convulsibility of the spinal cord has been attained, it is no longer necessary that the original lesion which gave rise to it should continue; for any impression which may then be received, either by the sentient nerves or by the brain, may lead to fresh convulsive attacks. Yet it is found as a rule that even then the convulsions will habitually start from the seat of the original

injury, and only gradually become generalised. As soon as the medulla oblongata has become implicated, the convulsions appear co-ordinated and bilateral, owing to the numerous nervous anastomoses which exist in that organ, and to the different nuclei of innervation being united with each other by commissural fibres.

Exaggerated impressions transmitted to the spinal cord by *sympathetic nerve-fibres* may have a similar effect. It is in this way that worms, certain forms of indigestion, renal and biliary calculi, will occasionally give rise to convulsions.

Finally, there are *cerebro-spinal* or *central* convulsions, caused by morbid irritation of the great nervous centre of motor power itself, which, as we have seen, extends from the spinal cord to the cerebral hemispheres.

Convulsibility of the motor centre may be produced by disease or injury at any portion of its course; and the convulsions are in this case generally confined to those parts which are under the immediate influence of the affected portion of the motor centre. A faulty constitution of the blood is the next cause, and where this is encountered the convulsions are liable to be general. Anæmia in children has a most powerful influence in producing general convulsions; the retention of excrementitious matters in the blood, such as urea, bile, and carbonic acid, leading to uræmia, cholæmia, and asphyxia, acts in a similar manner; and so does the introduction into the system of certain vegetable and mineral poisons, more particularly strychnia, brucia, picrotoxia, and lead. Elevation of the temperature of the blood in some febrile diseases, especially scarlatina, quinsey, and erysipelas, may also act as an exciting cause, provided there is a neuropathic tendency in the system. There may finally be a habitual constitutional state of undue excitability of the motor centre: the *neuropathic constitution*, which is either hereditary or acquired, may exist without structural lesions perceptible to our senses, and may, without any apparent cause, give rise to convulsions.

This convulsibility of the motor centre is frequently inherited from one or both parents; and it is not indispensable for this that there should have been convulsions in either of the parents, but simply a definite nervous affection of some

kind. Hysteria of the mother, and epilepsy and drunkenness of the father, are probably amongst the most potent and frequent causes of convulsions in infants. Drunkenness of one or both parents at the moment of conception is believed powerfully to predispose the offspring to convulsibility. The entire period of intra-uterine life is likewise influential; sudden shocks, painful emotions, grief, anxiety, privations, and fatigues of the mother, disturb the nutrition of the nervous system of the fœtus, and lead to convulsibility, imbecility, and epilepsy of the infant. Tubercular consumption of either of the parents is likewise an efficient cause, as such children are ill-nourished, flabby, feeble, and unduly timid and excitable.

Given a state of convulsibility of the motor centre, any impression, partaking of the nature of a moral or physical shock to special or general sensibility, whether perceived or not perceived, may give rise to a discharge of nervous force, or a *nerve-storm*, which may be local or general; and when the convulsibility has reached a high degree, such a storm may even break out without any apparent exciting cause at all. The nervous centre may then be aptly compared to a storm-cloud, from which lightning may be drawn forth either through external collision or an excessive internal charge.

The *emotional centres* of the brain are likewise of influence in the production of convulsions; fright, terror, prolonged grief and anxiety, a sudden impression on the imagination or memory, may produce a seizure. In such cases, however, we find as a rule that the convulsions affect more the sphere of the cerebral than of the spinal nerves, and that the parts chiefly affected are the face, eyes, and larynx.

As the most intimate connection exists between convulsions and a bloodless condition of the brain, I shall now proceed to consider this latter subject in its various bearings.

Cerebral Anæmia.

That the quantity of blood in the brain and its membranes is subject to considerable variations, is no longer doubtful at the present time. Two Scotch physicians, Kellie and Monro, have denied that such could be the case, provided the brain

and its membranes were in their normal condition. They thought the brain incompressible through being enclosed in rigid walls, and only liable to congestion or anæmia if there was previous wasting or hypertrophy of the organ. We now know that the brain is not only somewhat compressible, but that variations in the quantity of blood are also rendered possible by the vascular connection between the inner and the outer surface of the skull, and more particularly by means of the cerebro-spinal liquid, the quantity of which is in inverse ratio to the empty or loaded state of the cerebro-spinal blood-vessels. Thus we find this liquid largely increased in hydrocephalus where the brain is anæmic, while it is almost entirely absent in cerebral hyperæmia or determination of blood to the head. Most important of all, Donders has directly observed cerebral circulation through an opening in the skullcap of animals, which he closed by means of a watchglass; and seen considerable variations in the diameter of the bloodvessels of the pia mater, which he noticed to become dilated during expiration. It is reasonable to assume that in children, where the bones of the skull are not yet completely ossified, and the sutures open, congestion or anæmia may even be more readily brought about than in adults.

Sir Astley Cooper and Marshall Hall had already been struck by the resemblance of the phenomena observed after sudden and abundant hæmorrhage, with the fits of epilepsy and eclampsia; but it was Kussmaul and Tenner[1] who, by a masterly experimental investigation of this subject, showed that such attacks are invariably caused by rapidly induced cerebral anæmia. They employed vigorous, non-anæsthetised cats, dogs, and rabbits, and caused anæmia of the brain either by bleeding them to death, or by ligaturing both carotid and subclavian arteries—the latter, because the vertebral arteries arise from the subclavian. The invariable result was loss of consciousness and general epileptic convulsions, with involuntary evacuation of the urine and fæces. There was at first tonic rigidity, which was soon followed by clonic convulsions; and these were occasionally so violent as to throw the animal over the shoulders

[1] 'Untersuchungen über Ursprung und Wesen der fallsüchtigen Zuckungen, Moleschott's Untersuchungen,' vol. iii. 1857.

of the operator. Where the ligature was used, the most violent convulsive fit could at once be arrested by removing the same from only one carotid. It was also found in these experiments that the functions of the spinal cord and medulla oblongata continued for some time after that of the brain had been annihilated; for respiration and reflex excitability persisted during the coma, and the animal could be restored to life as long as the heart's action was not entirely suppressed. That there was true cerebral anæmia in these cases was also shown by Donders's watch-glass experiment.

If cerebral anæmia was only partial, by leaving one carotid or vertebral artery open, the animal became drowsy, feeble, and delirious, but was not convulsed. Compression of the carotid arteries in men had nearly the same effect as ligature of the same in animals.

Kussmaul and Tenner also showed that hyperæmia of the brain does not cause any convulsions. In order to prove this they first divided the cervical sympathetic in rabbits, and afterwards tied both the external and internal jugular veins, with the result that no convulsions appeared.

A cross experiment which was intended to produce cerebral anæmia and convulsions by Faradisation of the cervical sympathetic, succeeded only once, in a rabbit. They first tied one carotid and both subclavian arteries, and then faradised the sympathetic of that side where the carotid had not been tied. The fundus of the eye became suddenly pale, the pupil dilated so much that the iris disappeared, the neck was drawn back, and violent convulsions were produced. On discontinuing the Faradisation, the fundus of the eye became red, the convulsions ceased, and the pupil contracted. That these phenomena should not have been produced in other experiments is explained by the circumstance that only a portion of the vasomotor nerves of the brain courses through the cervical sympathetic.

Nothnagel has succeeded in causing epileptiform seizures in animals by irritation of the peripheral nerves. In this way contractions of the cerebral arterioles and consequent anæmia was caused by reflex action.

Anæmia of the spinal cord does not cause convulsions. The

cord therefore serves only as conductor, and the origin of the convulsions is in the pons and the medulla oblongata.

Cerebral anæmia may be produced by injury of a large artery, the rupture of an aneurism, flooding after childbirth, and ligature of the carotid artery, which has been performed for the cure of aneurism and of tic douloureux. In the few cases in which both carotids have been tied, the patients have died soon afterwards from cerebral anæmia. This is also induced, not only when the brain receives too little blood, but also when its composition is altered, and more particularly so when the number of red corpuscles is considerably diminished. Interstitial nutrition of the brain must be energetic from one instant to another, in order to allow a proper function of the organ to be carried on; for no tissue is more easily vulnerable than cerebral tissue. Without a continuous supply of oxygen such interstitial nutrition is impossible; and as the red corpuscles are the carriers of oxygen, any considerable decrease in their number must have an unfavourable influence on the condition of the brain. Instances of this are seen in starvation, where the quantity of the blood altogether is diminished; in anæmia, where the number of red corpuscles is diminished; and in ischæmia, where some impediment to circulation prevents the cerebral arterioles from receiving a proper supply of blood. It is by this latter that sudden death from cerebral anæmia may be caused in convalescents from acute diseases, such as yellow fever. In cases of this kind the blood accumulates in the lower extremities and the abdomen, when the patient, who has been reclining for a long time, suddenly gets into the erect position. The collapse on tapping for ascites, or an ovarian cyst, has to be similarly explained. By removing a large quantity of liquid the intra-abdominal pressure is suddenly reduced; the veins thus receive an unusually large supply of blood, and as they are enfeebled by long-continued compression, it accumulates in them; the arteries therefore receive too little blood, and cerebral anæmia is the result, the *vis à tergo* being insufficient to determine a proper supply of arterial blood to the head. A similar result is caused when other organs suddenly receive an unusually large quantity of blood, as the leg during the application of Junod's cupping-boot. In certain exhaustive diseases, such as

dysentery, chronic nephritis, cancer, phthisis, saturnine and mercurial poisoning; in protracted suppuration; in typhoid fever and small-pox, where the intestinal ulceration and the pustules of the skin impoverish the blood; in women who have borne a number of children in rapid succession and suckled them all; and where the intracranial space is much reduced by the effusion of serum, the extravasation of blood, or tumours, the like result may be brought about. Certain drugs, such as ergot and quinine, are believed to have a similar effect; what is less doubtful is that some diseases of the heart have the tendency to cause cerebral anæmia. Thus we find that in fatty degeneration of the heart and myocarditis, there is not sufficient pressure exerted by the muscular substance of the organ to carry sufficient blood into the carotids; also where the aortic ostium is narrowed and the valves do not close; and compression of the umbilical cord during labour has the same effect on the fœtus.

Cerebral anæmia is on the whole most frequent in infancy and old age. In the aged it is habitually caused by atheromatous degeneration of the cerebral arteries. These are reduced in diameter by the deposit, and the circulation in the brain is thereby impeded. This is still further aided by the rigidity of the arterial coat, for Marey has shown that liquids are propelled more slowly in rigid than in elastic tubes; and by the insufficient strength of the heart's action, which is the rule in the aged.

In infants and children, whose more yielding skull adapts itself more readily to changes in the quantity of blood, the most potent cause of cerebral anæmia is improper feeding. In foundlings and the children of the poor this is habitual, and generally leads to diarrhœa, which in a short time causes general and cerebral anæmia. Treatment by depressant medicines may have the same effect. The symptoms which are observed under these circumstances strongly resemble those of acute hydrocephalus or tubercular meningitis; and Marshall Hall has therefore proposed the term 'hydrencephaloid' for these cases. There is a stage of excitement followed by stupor. The children are restless and frightened, scream, moan, and sob, are unduly sensitive to light, noise, and touch, and in a general state of convulsibility. The skin is hot, the face red, the pulse

frequent, and the pupil constricted. This stage is more or less rapidly followed by prostration, pallor of the face, insensibility, dilatation and immobility of the pupils, and anæsthesia of the nerves of special sense. The respiration becomes difficult and intermittent, the pulse imperceptible, and coma is followed by death. There are few diseases in which the influence of treatment is so marked as in this; for if in the first stage the condition be treated as inflammatory, by lowering remedies, the fatal issue is considerably accelerated; while treatment directed towards arresting the diarrhœa and restoring strength, is often followed by excellent results in apparently desperate cases. Recovery is under these latter circumstances wonderfully quick; the pulse reappears, respiration becomes regular, the skin warm, the pupil contracts, and in a few days health is restored.

In adults, where cerebral anæmia is rapidly produced, as in flooding after labour, or after ligature of the carotid artery, the symptoms are as follows: everything suddenly appears black before the eyes of the patient; there is tinnitus and vertigo; the pupils dilate; a feeling of faintness, with sickness and vomiting supervenes; there is tremor, general convulsions, and loss of consciousness. The skin appears white, the pulse is small and hardly perceptible, respiration retarded and intermittent; and death is occasionally preceded by delirium.

There is one form of anæmia of the brain which may occur suddenly without any loss of blood having taken place, and without any of the other causes which have just been mentioned. This is *vasomotor anæmia*, arising from fright or shock. Slighter painful emotions cause pallor and chilliness of the skin, particularly of the face, owing to sudden vascular contraction; but where the emotional disturbance is severe, a spasm is produced in the vasomotor nerves of the pia mater sufficiently intense to cause tetanus of the cerebral arterioles, in consequence of which these are entirely emptied. The brain suddenly becomes bloodless: there is vertigo, fainting, loss of consciousness, and sudden death, unless the contraction is quickly relieved. This is the physiological explanation of the condition which was by the older writers called *nervous apoplexy*. In many cases, however, the spasm of the cerebral arterioles is quickly followed by relaxation, and hyperæmia then takes the place

of anæmia. The vertigo and loss of consciousness are succeeded by excitement and delirium; and the patient only recovers after the circulation of blood in the brain has become equalised.

When cerebral anæmia is slowly developed, the symptoms are quite different. In the aged, the principal symptom is giddiness. The sight becomes dim, the patient is seen to stagger and calls for support in order not to fall; he soon recovers himself, but the vertigo reappears from the least or apparently no cause, and that not unfrequently several times a day. Emotions or muscular efforts are particularly apt to induce such attacks. At the same time the temper is irritable, there is intolerance to light and noise, and general hyperæsthesia of the special senses, with delusions, and greatly impaired memory. The speech is slow, there is great indifference to things in general, and senile dementia is apt to follow. In many persons of this kind there is habitual drowsiness; they rally on being spoken to, and answer questions, but quickly relapse into somnolence, and sleep the better part of the day as well as night. The muscular system is very feeble, the heart's action insufficient, the pulse much accelerated by the slightest mental or bodily exertion, and very compressible. Sometimes drowsiness alternates with a delirium resembling that of inanition.

Where anæmia implicates more particularly the posterior lobes of the brain, the symptoms are rendered somewhat peculiar, in consequence of the vaso-motor connection of the bloodvessels of these lobes with those of the abdominal viscera. While the anterior and parietal lobes are supplied by the internal carotid arteries, the posterior lobes are fed by the basilar, which is in intimate communication with the splanchnic nerves, the cœliac ganglion and its branches. Anæmia of the posterior lobes may thus be accompanied with disturbances of the abdominal circulation, loss of appetite, congestion of the liver, accumulation of fæces in the large bowel, disease of the uterus, supra-renal capsules, and other viscera; and its chief clinical symptom appears to be melancholia, by which we know many of the diseases mentioned to be so generally accompanied.

After death from cerebral anæmia the sinuses of the dura mater and the veins of the pia mater are seen to be empty, or

nearly so. What little blood there is appears thin and watery, and watery coagula of fibrine are found. The grey matter of the brain is so pale as to resemble the white, and the white matter has a shining milky appearance, and is tough and dry. Where there is no actual brain disease, the anæmia is mostly general; but where there are other diseases, such as a fresh clot, circumscribed encephalitis, or a tumour, local hyperæmia may be present. The ventricles and the meshes of the pia mater contain more serum than usual.

Practically we have to distinguish three principal forms of eclampsia, which have this in common that there is loss of consciousness, tonic or clonic involuntary muscular contractions, and great danger to life. These are:—

1st, the eclampsia of children, which is owing to the predisposing influence of the infantile period of life;

2nd, the eclampsia of pregnant and parturient women, which is owing to the influence of pregnancy and child-birth, in connection with Bright's disease of the kidneys; and

3rd, the eclampsia produced by the influence of certain poisons, such as lead, carbonic oxide, nicotine, picrotoxine, etc.

1. *Infantile Eclampsia.*

The attack of eclampsia in children is either sudden, or preceded by symptoms showing undue excitability on the part of the nervous system. The child is cross, self-willed, passionate, and fidgety; its facial expression is weary and restless at the same time; sleep is disturbed by grinding of the teeth and sudden screams; the breath is short; there is somnolence in the daytime, and muscular twitches occur, particularly in the face, producing the sardonic laugh, from convulsibility of the zygomaticus major and platysma myoides muscles. Then the fit of eclampsia breaks out suddenly:—the child utters a scream, and loses consciousness; the physiognomy becomes vacant; the face distorted; the eyes are drawn back into the orbit and rolled upwards; there is pallor, which is generally of short duration and succeeded by lividity, owing to the accumulation of carbonic acid in the blood, respiration being arrested by spasm of the glottis and rigidity of the respiratory muscles.

The face is sometimes perfectly black, and the veins of the neck appear filled to bursting. The neck is stiff; the head drawn to the side and a little backwards by the spasmodic action of the cleido-mastoid and trapezius muscles. The upper extremities are pronated, and the fingers strongly flexed; the lower extremities extended and rotated inwards or outwards. The spine is stretched, the chest-walls are rigid and immovable, the glottis closed, and the diaphragm and abdominal muscles in a state of tetanus. The limbs are generally cold, and the body hot. Fæces and urine are expelled involuntarily, and death may take place with almost lightning-like rapidity in the first attack. In most cases, however, after tonic rigidity of the whole frame has existed for a short time, a whistling inspiration is heard; the glottis opens again, the chest-walls move, a little air is admitted, the face loses its lividity; the spine bends, the head is seen to fall back with each period of muscular relaxation, but again drawn to the side by fresh muscular contractions; tracheal and bronchial mucus mixed with blood flows from the mouth. The blood comes either from the bitten tongue or from the mucous membrane of the cheeks, which is liable to hæmorrhage from excessive congestion and eventual rupture of the capillary vessels. The same cause may give rise to hæmorrhage in the brain and other organs, and hemiplegia with aphasia may therefore be the result. The violence of the convulsions is sometimes so great that the muscles are actually torn to pieces, and the bones become fractured or dislocated.

The attack generally lasts from two to five minutes. The whistling inspiration of the convulsed glottis, as it rapidly opens and closes, is followed by stertorous breathing, which after a time merges into tranquil respiration. The clonic convulsions are succeeded by a state of extreme prostration and relaxation of the whole body. Stupor is sometimes followed by a deep and restorative sleep, from which the little patient awakes apparently quite well. At other times, however, the first fit is rapidly followed by a second, and many more; and death takes place by the coma gradually deepening. The fatal result is then owing either to nervous exhaustion in consequence of too powerful discharges, or to deficient oxidation of the blood, in consequence of repeated attacks of asphyxia. This so disturbs

innervation that, although there may be no fresh convulsive seizures, yet the patient appears to die from gradual suffocation, i.e. an excess of carbonic acid in the blood, which there is not sufficient systemic energy to eliminate.

In such cases, where the attacks follow each other almost incessantly; where collapse and coma continue between and after the attacks; where pulse and respiration are considerably accelerated, we also find a great and rapid *increase of temperature*, which may attain 108° and even 109°, and which sometimes attains its maximum a few hours after death. Where the attacks chiefly affect the limbs, and do not interfere with respiration, they may last for hours and even days without becoming dangerous.

The general convulsions which affect the muscles of the face, neck and chest, as well as those of the limbs, and which arise from over-stimulation of the pons and medulla oblongata, are always bilateral; yet one side is often more severely affected than the other, so that the child, always drawn to the same side, may fall out of bed unless prevented. Partial convulsions of the limbs are often unilateral, but may also be bilateral. During these partial attacks the pulse often does not undergo any particular changes; while in general convulsions it is mostly quick, full, and dicrotous.

A series of attacks rarely lasts longer than a week. During all this time there may be a state of exalted excitability of the nervous system, as shown by jerky respiration, squinting, sardonic laugh, flexion of toes and thumbs, etc. Slight causes are sufficient to bring on a fresh fit, but as time progresses, the convulsible condition is gradually replaced by one of lowered excitability. The sequelæ are not unfrequently stammering, contractions of the muscles, imbecility, aphasia, paralysis, amaurosis, and finally true epilepsy, which may only become developed about the period of puberty.

Dentition has generally been accused as being the chief cause of convulsions in infants, but recent investigations do not confirm this notion. It is more in consonance with facts to say that in the first year of life, the brain of infants is extremely delicate, and grows very rapidly. The excitability of the cerebro-spinal centre is excessively great at this time, and slight

causes which would pass without effect in adults, are in infants sufficient to excite grave mischief. The period of first dentition and that of weaning are no doubt particularly liable to convulsive seizures; but these latter do not by any means cease when a tooth is through, nor can lancing the gums be considered the principal remedy for infantile convulsions.

Excessive cold and heat have a decided influence on the production of this disease. Just as on the battle-field a night-frost will give rise to many cases of trismus and tetanus amongst the wounded, the severe cold of winter is an important factor in the causation of infantile eclampsia. Exposure to night-air is particularly dangerous; and the children of the poor, who are more liable to be affected by the vicissitudes of weather, suffer in preference to those of the well-to-do classes, more particularly as the former are generally subject to exhaustive diarrhœa, brought on by chills and improper feeding.

The excessive heat of summer has an equally deleterious effect on many infants and young children. It is not necessary that they should be exposed to the direct rays of the sun, but simply that the temperature of the atmosphere should be very high. Indeed, the symptoms often commence at night, in the close, foul, hot air of a bedroom, in which several children sleep with closed windows. The principal initial symptom is, that the skin which has been perspiring freely, suddenly ceases to do so, and becomes dry and burning hot. This is followed by severe diarrhœa, and often speedily, at other times less quickly, by an attack of general convulsions and coma; sometimes death takes place in the attack of asphyxia, while in other cases the fatal result seems to be owing to syncope: the child, after an attack, starts up in his cot, gives a few convulsive gasps, the face appears blanched, the pupils are widely dilated, the pulse is imperceptible, and the little patient falls back dead. Again, in other cases death takes place by the coma gradually deepening. In these cases we must assume that the superheated blood produces a paralytic state of the sweat-glands, most probably through its poisonous influence on the vaso-motor centre in the medulla oblongata. While, therefore, the influence of external heat continues, there is no longer any cooling of the surface, to counterbalance it by perspiration and the evaporation

of the sweat; the temperature of the blood must therefore rise, and this rise causes a rapid destruction of the red blood-corpuscles, with accumulation of effete matters in the blood. Cases of this kind are not unfrequently looked upon as such of cholera infantum, or enteritis, connected in some mysterious manner with dentition; and the children are treated by lancing of the gums, and astringents for checking the diarrhœa; while the only chance of the child's life lies in a successful endeavour to restore the action of the skin, which should be done by placing the little patient into a cold bath, afterwards 'packing' him in a wet sheet, and administering small doses of aconite, or Warburg's fever tincture. The sudden relief which is given by these measures, in arresting convulsive seizures, procuring sleep, and checking the diarrhœa, amply proves that the pathology of the cases as just given is the correct one.

The acute exanthemata, and more especially scarlet-fever, which have been much accused, have less to do with the production of eclampsia than is generally believed. They mostly occur at a somewhat later period of life than eclampsia, which we have seen to be incomparably more fatal in the first year than in subsequent periods of life. Of small-pox, there were in 1874, only 306 fatal cases in the first year of life; of measles 2,607, and of scarlet fever 1,436; while the numbers of these diseases altogether were: 2,162 for small-pox, 24,922 for scarlet fever, and 12,255 for measles. Convulsions, however, do occur in the commencement as well as in the later stages of scarlatina, and in the latter case are generally owing to acute nephritis, and associated with dropsy. Remote sources of irritation, such as renal or vesical calculi and tickling of the soles, may also reflexly lead to convulsions. In suckling children mental emotions of the mother or wet-nurse; in older children emotions of their own, such as jealousy or fright, may act as exciting causes.

The post-mortem appearances are in the majority of cases due rather to the mode in which death has taken place, than to any lesion specially characteristic or causative of eclampsia. They are generally those of death from asphyxia, viz. a large quantity of dark blood in the brain and meninges. This has often been considered the cause of the disease, while it has

really been only an effect. In many cases the results of the post-mortem examination are entirely negative.

2. *Puerperal Eclampsia.*

It is at present an open question whether puerperal eclampsia should continue to be considered as a special disease of itself, or simply be brought under the heading of Uræmia. There can be no doubt that in the vast majority of cases the convulsions of puerperal eclampsia are owing to the retention of urea in the blood, and that the impure blood, by its influence upon the vasomotor nervous centre, leads to that contraction of the arterioles which causes sudden and excessive anæmia of the brain, and as a consequence of this, convulsions. Yet the puerperal state causes certain modifications in the affection which render it for the present desirable to retain the denomination of puerperal eclampsia.

Not all cases of convulsions occurring during the later periods of pregnancy and childbirth belong to this class; for parturient women are subject to hysterical and epileptic attacks, and capillary apoplexy and meningitis have likewise been known to give rise to convulsions in that state. Puerperal eclampsia can only be said to exist when convulsions of a somewhat peculiar character occur, during the last few months of pregnancy, or during labour, in women not habitually hysterical or epileptic, but who are generally, although not invariably, subject to Bright's disease of the kidneys, and consequent blood-poisoning.

The attack of puerperal eclampsia closely resembles the attack of epilepsy. There is loss of consciousness and convulsions, followed by coma; yet some differences may habitually be noticed. There is generally no initial cry or scream; consciousness, which in epilepsy is lost at the very commencement of the fit, is more gradually lost, and completely so only when the attack reaches its acmé; and where a series of attacks takes place, consciousness is not at all recovered from before the patient dies, or until after delivery has taken place, when the attacks cease completely. Stupor may, indeed, continue for days; and is, as a rule, so deep that the mother, on recovering

herself, is unaware of having been delivered, and refuses to acknowledge the child as her own.

In other respects the attack of eclampsia is epileptiform; there are tonic and clonic convulsions; the tongue is bitten; there is foam at the mouth; in bad cases the urine and fæces are voided involuntarily, and there may be delirium and maniacal excitement, as well as paralysis and anæsthesia after the attack. The fit lasts from two to fifteen minutes, and begins either without any warning, or after such premonitory symptoms as the following: restlessness, headache, sickness, squinting, and partial convulsions of the facial muscles. Sometimes there is only one attack followed by coma, after which the patient recovers; in other cases a series of attacks takes place in rapid succession without any recovery of consciousness.

To these symptoms on the part of the nervous system are in most cases added indications of nephritis, viz., œdematous swelling of the eyelids, ankles, and labia; albumen and tube-casts in the urine; and in severe cases uræmia, i.e. the presence of carbonate of ammonia in the blood. The latter signs, taken together with the premonitory symptoms just mentioned, may indeed enable the physician in some cases to predict the occurrence of puerperal eclampsia.

It is unquestionable that convulsions are more common in the uræmia of pregnancy and the puerperal state than in other forms of uræmia; and this must be ascribed to the exalted state of reflex excitability at that period of female life. Physical and emotional causes are then alike active in producing an excess of irritability, which is favourable to the production of convulsions.

Scanzoni and some other high obstetric authorities think that the 'albuminuria' of pregnancy is owing to venous congestion produced by the attack of eclampsia, which latter they consider the primary disease. If Bright's disease be present, they look upon it as a complication of the eclampsia, the real causes of which they seek in increased nervous excitability and certain mechanical influences, such as rigidity of the neck of the womb, transverse position of the fœtus, certain obstetrical operations, and profuse hæmorrhage from the uterus. But this view is most certainly not borne out by the facts of the case.

In the first place, we have to notice the physiological fact that removal of both kidneys in animals gives rise to uræmia and convulsions. Secondly, it is contrary to fact that convulsive attacks tend to produce albuminuria, because even after a long series of epileptic attacks no albumen is found in the urine. Thirdly, the disease is not mere albuminuria, or passive transudation of albumen from congestion, but real nephritis, as shown by the microscopic appearances of the urine during life, and of the kidneys after death. Fourthly, this nephritis is almost invariably prior to the convulsions; for although in some well-observed cases albumen has been absent from the urine before a fit, and present after one, it seems that œdema has always preceded the fit. Now it is well known that albumen in the urine is not always the first symptoms of nephritis; for in certain cases of scarlatina dropsy has been observed before the albuminuria.

The cause of the appearance of nephritis before and during parturition is still obscure. It has been assumed to be owing to pressure, but this view is surely erroneous, as the nephritis occurs sometimes in the earlier months of pregnancy, when there is little pressure; is absent in the immense majority of cases of parturition, where there is a great deal of pressure; and likewise in cases of ovarian dropsy, where the pressure is often greater than in pregnancy. Dr. Galabin thinks both the nephritis and eclampsia due to the circulation of some irritating material in the blood; and in the absence of any other reasonable explanation we may for the present accept this as probably the true view. It might be supposed, seeing the very large number of women who bear children, that nephritis should occasionally appear in them without any other cause than one of those to which it is usually owing, viz., cold, excess in drinking, etc.; but that the nephritis is truly connected with pregnancy and parturition is shown by the circumstance that the disease generally vanishes after the lying-in period.

Puerperal eclampsia is not frequent. Professor C. Braun has in 24,000 consecutive cases of which notes were taken at the Vienna Maternity, seen only 44 cases of this affection, which would give one case to 544 births; and in the practice of other observers its occurrence appears to be still more rare.

The death-rate is generally put at 33 per cent. In England and Wales there died of puerperal convulsions, in 1874, 514 women. The number of births in the same year was 854,956, which would give one fatal case to 1,675 births. These numbers tally remarkably with those of Professsor C. Braun; for as one case in three proves fatal, it is seen that one case of eclampsia would occur in England and Wales in 558 births, the number for Vienna being 544.

The disease appears to be more frequent in the first than in subsequent pregnancies, and in young, robust, and full-blooded women more so than in weak and elderly persons. Those who have once suffered from it seem to be liable to further attacks in subsequent confinements. Amongst Braun's 44 cases, 32 occurred between 15 to 25, and the remaining 12 cases between 25 and 40 years of age.

Where eclampsia comes on during pregnancy, the life of the fœtus is more endangered than where it appears during parturition. This danger is also greater in the first periods of labour, more especially where the pains are tardy, and the os does not dilate well, than in the later ones; for the labour-pains are generally increased by the convulsive attacks, and these latter mostly cease after delivery. The convulsions, however, do not correspond in their occurrence with the labour pains. Puerperal eclampsia is often found to co-exist with a narrow pelvis, an excessive quantity of liquor amnii, and twin-births; and it predisposes to puerperal fever, and other puerperal affections.

It is a singular fact that in all forms of uræmia, and even in that in which the patients become affected with convulsive seizures, the temperature is considerably lowered from the very beginning; and as the symptoms of uræmia become more strongly marked, the temperature continues to fall, and reaches a minimum—82°·5—previous to death. In the uræmic eclampsia of pregnancy and parturition, on the other hand, we notice absolutely the reverse. Quincke[1] and Bourneville[2] have shown that the temperature rises rapidly as soon as the convulsive attacks commence; that there is a further increase with every fresh fit; and that the maximum, amounting in some

[1] 'Berliner klinische Wochenschrift,' 1869, No. 29.

[2] 'Etudes cliniques et thermométriques,' &c., part ii. Paris, 1873.

cases to 110°, is attained just previous to or soon after death. In cases that are to end favourably, on the contrary, the temperature falls as the fits cease and the coma diminishes; and it eventually again reaches the normal standard. We are at present utterly unable to explain these extraordinary differences in the temperature in two pathological conditions which otherwise resemble each other so very closely.

3. *Eclampsia from Poisoning.*

Certain poisons, when habitually introduced into the system, give rise to a most dangerous form of eclampsia. Amongst these lead is the most important. Saturnine epilepsy, as this affection is also sometimes called, is very rare; it only occurs in persons who have for a considerable time shown symptoms of lead-poisoning, such as colics, arthralgia, paralysis &c., and in whom the system is thoroughly saturated with the metal. The eclampsia of lead is in no way connected with uræmia or albuminuria, but occurs in those habitually exposed to the influence of lead, more especially house-painters and compositors. The attacks last longer, and are more frequent than those of epilepsy, to which they otherwise resemble, and generally end fatally. Sometimes there are as many as thirty in one day. The temperature under these circumstances rises rapidly. Consciousness is rarely regained, and death takes place either from asphyxia, or with the symptoms of apoplexy, within a few days from the outbreak, of the convulsions. In a few cases where the patients rallied, they remained incapable of mental or physical exertion, and in a somnolent condition, and they were ultimately carried off by a fresh outbreak of the disease.

That it is really the lead which causes these symptoms is shown by physiological experiments, in which dogs fed with subacetate of lead ultimately perished with eclampsia, and amaurosis. In these animals the kidneys remained healthy; there was no carbonate of ammonia, and hardly any urea in the blood; but lead was found in the brain, which was greatly anæmic. Lead, by its astringent effects on the bloodvessels, causes them to be constricted to such an extent that ultimately

very little blood can pass through the cerebral arterioles. These results are confirmed by those of post-mortem examinations of men who have died of saturnine eclampsia, and in whom the cerebral substance was found quite bloodless, and the ventricles empty.

Certain other poisons cause attacks of eclampsia, viz. carbonic oxide, carburetted hydrogen, hydrocyanic acid, coniine, nicotine, and picrotoxine; but these, from their rarity, are devoid of practical importance.

Convulsive seizures which occur at the commencement of the acute exanthemata are probably more owing to the high temperature of the blood than to the poison contained in the same. Such fits also occur when nephritis sets in in the course of scarlet fever, or pyæmia in the progress of small-pox, in erysipelas of the face, and other inflammatory conditions in the neighbourhood of the head, in which considerable elevation of the body-heat is observed.

CHAPTER III.

APOPLEXY.

Not fewer than twelve thousand persons die annually in England and Wales of apoplexy; and we may assume that at least twice as many people living in these parts suffer from the effects of it. It constitutes, after convulsions, the most fatal of all diseases of the nervous system, carrying off year by year, with unerring certainty, more victims than either paralysis, epilepsy, or insanity. If we consider it only numerically, therefore, apoplexy must be ranked with the most important diseases which we are called upon to treat in practice; but as it threatens the life chiefly of the aged and middle-aged, that is, practically speaking, the heads of families, its importance appears by this circumstance to be considerably increased, for the social position of entire families is often completely altered by its occurrence.

The following table shows the exact number of deaths from apoplexy which have been registered during six periods of five years each:—

Periods of five years	Deaths from Apoplexy	Percentage of Deaths from all Nervous Diseases	Percentage of Deaths from all causes
1838–42	28,316	13·49	1·63
1843–46	Not registered	—	—
1847–51	39,321	16·39	1·94
1852–56	41,681	16·15	2·00
1857–61	43,414	15·97	1·99
1862–66	50,691	17·30	2·11
1867–71	55,158	17·85	2·20
Total of thirty years .	**258,581**	**16·36**	**2·00**

Dr. Lidell,[1] who has extracted the deaths from apoplexy in the City of New York from the Annual Reports of the Metropolitan Board of Health of that city, has found the mortality from apoplexy to be 1·40 per cent. of the entire mortality from all causes. Supposing the same diseases to be registered under the heading 'apoplexy' in New York as they are in this country, the percentage for the American city is considerably less than that for England and Wales.

Age has a most important influence on the production of apoplexy, as is shown in the annexed diagram (p. 84). We see from this that *apoplexy, although not uncommon in infants, is essentially a disease of old age.*

In the infantile period of life it is chiefly the first year which is liable to apoplexy, after which there is a decided fall. The curve shows

126	deaths	in	the	1st	year of life,
38	„	„	2nd	„	„
26	„	„	3rd	„	„
26	„	„	4th	„	„
13	„	„	5th	„	„

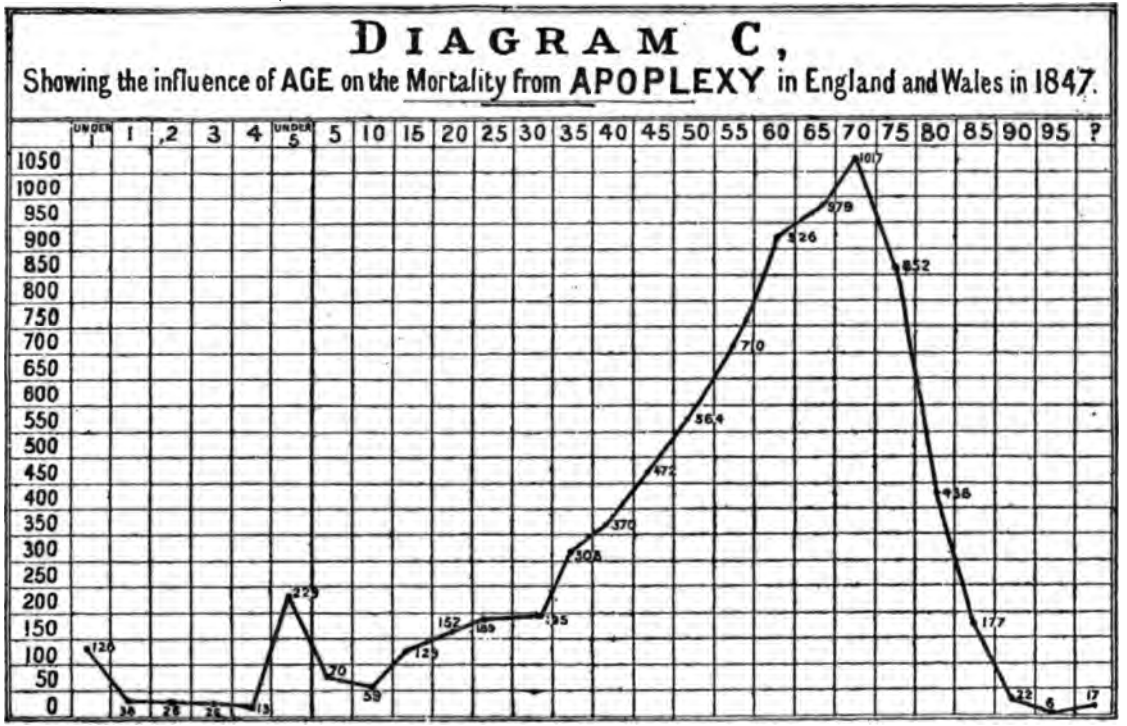
The entire number of deaths for the first period of five years is therefore 229, and this considerably exceeds that of the immediately succeeding periods up to 35 years of age, when the disease again becomes more largely fatal. The minimum is reached at ten; after that there is a rise which is at first slow, but gradually becomes more rapid. The maximum is reached at seventy, and then there is another fall, but at no time of life is there any actual cessation. The following are the exact numbers:—

Under	5	years of age	.	.	229	deaths.
From	5	to 9	years of age	.	70	„
„	10	„ 14	„	.	59	„
„	15	„ 19	„	.	129	„
„	20	„ 24	„	.	152	„

[1] 'A Treatise on Apoplexy.' New York, 1873. P. 30.

DIAGRAM C,

Showing the influence of AGE on the Mortality from APOPLEXY in England and Wales in 1847.



The top line of numbers denotes the periods of age at which the deaths have taken place.
The side line of numbers indicates the numbers of deaths at the different periods of life.

From 25 to 29	years of age .		183	deaths.
„ 30 „ 34	„ .		195	„
„ 35 „ 39	„ .		308	„
„ 40 „ 44	„ .		370	„
„ 45 „ 49	„ .		472	„
„ 50 „ 54	„ .		564	„
„ 55 „ 59	„ .		710	„
„ 60 „ 64	„ .		926	„
„ 65 „ 69	„ .		979	„
„ 70 „ 74	„ .		1,017	„
„ 75 „ 79	„ .		852	„
„ 80 „ 84	„ .		438	„
„ 85 „ 89	„ .		177	„
„ 90 „ 95	„ .		22	„
Over 95	„ .		6	„
?	„ .		17	„

Considering how few people are alive after 74, the mortality from apoplexy appears very large indeed in the latest periods of life. This is no matter of surprise if we consider that apoplexy in its most frequent form, viz., that which is produced by cerebral hæmorrhage, is essentially a disease of vascular decay, which is most largely encountered in advanced age. Deaths from this form of apoplexy are rare before forty years of age; and it is therefore fair to assume that those deaths which occur previous to that period are chiefly induced by meningeal hæmorrhage, cerebral hyperæmia, encephalitis, acute alcoholic intoxication, and embolism of cerebral arteries. In the later periods of existence, apoplexy is mostly owing, besides to cerebral hæmorrhage, to that form of meningeal hæmorrhage which is connected with pachymeningitis (hæmatoma of the dura mater).

Dr. Lidell's tables of apoplexy in the city of New York show on the whole the same proportion of the different periods of life. In order to facilitate a comparison between the mortality from the same disease in New York and in England, I have put the figures side by side in the following table:—

Deaths from Apoplexy	New York, 1867–69	England and Wales in 1847
Under 10 years of age . .	68 deaths	299
From 10 to 19 years of age .	15 „	188
„ 20 „ 29 „ .	60 „	335
„ 30 „ 39 „ .	146 „	503
„ 40 „ 49 „ .	177 „	842
„ 50 „ 59 „ .	187 „	1,274
„ 60 „ 69 „ .	209 „	1,905
„ 70 „ 79 „ .	142 „	1,869
„ 80 „ 89 „ .	32 „	615
Over 90 years of age . .	2 „	45

These figures show a very close analogy, and go far to prove that the production of apoplexy in America obeys, as far as age is concerned, the same laws which obtain in this country. The first period of life is in both countries more fatal than the second; after the second period, an uninterrupted rise takes place until the decade from sixty to seventy; and after that there is an uninterrupted fall to the end. It is however to be remarked that, while for New York the difference between the seventh and eighth decennium of life is considerable, it is very slight for England and Wales, which probably means that longevity is greater in England than in the States.

While, therefore, age is found to have a most powerful influence in the production of apoplexy, my researches tend to show that *sex* has, at least as far as this country is concerned, only little influence upon it. Up to the present time it has been generally assumed that males are greatly more liable to die from this disease than females; but it will be seen from the following

Mortality of Males and Females from Apoplexy in England and Wales.

	Males	Percentage	Females	Percentage
1847–51	19,800	8·26	19,521	8·21
1852–56	20,756	8·06	20,925	8 06
1857–61	21,508	7·93	22,106	8·13
1862–66	24,704	8·47	24,987	8·57
1867–71	27,565	8·92	27,593	9·00
Average of 25 years .		**8·33**	—	**8·40**

table that in England and Wales the sexes die in nearly equal proportion of apoplexy, and that the slight excess which is found to exist is not on the side of males, but on that of females.

These numbers have to be considered in connection with the circumstance that the female population of England and Wales constantly exceeds the male by more than half a million, and that the mortality of males constantly exceeds that of females by nearly 20,000. Thus, in 1874, matters stood as follows :—

	Males	Females	Excess of females
Population living .	11,512,956	12,135,653	622,697
Deaths from Apoplexy . . .	6,420	6,428	—
General mortality .	272,178	254,454	—
Percentage of mortality from Apoplexy to general mortality . .	2·36	2·53	—

It therefore appears that *apoplexy is more fatal to females than to males, to the extent of* ·17 *of the entire mortality from all causes.*

Dr. Lidell's tables show a preponderance of the male over the female sex in New York as far as mortality from apoplexy is concerned; but his cases amount altogether only to 1,038, and extend over three years only, while mine are infinitely more numerous, and extend over twenty-five years; so that my evidence, at least as far as England and Wales are concerned, must be considered irresistible.

The general agreement of continental authors as to the greater prevalence of apoplexy in men may possibly have its foundation in the different habits of English and continental women. I shall have to show in a subsequent chapter that delirium tremens is much more frequent in the female sex in England than elsewhere; and as some forms of apoplexy are unquestionably owing to intemperance, it may be assumed that the greater addiction to drink of females of the lower classes in this country compared to others increases their liability to

apoplexy. In no other capital of Europe are drunken women as frequently seen as in London; and if the excesses publicly committed by them on Bank Holidays correspond to what takes place more privately in ordinary times, their great liability to apoplexy is easily accounted for.

I now proceed to analyse the pathological conditions which give rise to death from apoplexy. Apoplexy[1] means in general medical parlance the condition in which a person has more or less suddenly lost his consciousness, sensibility, and motility while respiration and the heart's action continue. It is thereby distinguished from *syncope*, in which the heart's action is suspended, and from *asphyxia*, in which respiration has been arrested. Apoplexy was formerly believed to be a disease of itself, but modern investigations have shown that it is merely a symptom of disease, and that it may be produced by the following pathological conditions:—

1. Hæmorrhage into the brain.
2. Hæmorrhage into the membranes of the brain.
3. Hyperæmia of the brain, or determination of blood to the head.
4. Embolism of cerebral arteries.
5. Acute alcoholic intoxication.
6. Acute intoxication by opium and other narcotic poisons.
7. Sunstroke.

In the following I shall analyse these diseases in their connection with apoplexy, and begin with the most important of them, viz. hæmorrhage into the brain.

1. *Cerebral Hæmorrhage.*

This is by far the most frequent cause of apoplexy; but in spite of its great importance its anatomical and clinical features have only of late been more accurately ascertained. The old ideas about its being due to the rupture of an atheromatous bloodvessel are still found in the text-books; and many highly significant symptoms, which are not only of great pathological interest, but also of considerable value for determining the prognosis of the affection, are not yet familiar to practitioners,

[1] From ἀπὸ and πλήσσω, or πλήκτω, to strike, to knock down.

from attention not having been sufficiently directed to their occurrence and meaning.

Cerebral hæmorrhage is produced—

1. *By rupture of the large cerebral arteries*, such as the middle cerebral and basilar, after these vessels have undergone ordinary aneurismal dilatation. (*Vide* Chapter XI.)

2. *By rupture of the capillary vessels*, which is generally caused by injury to the head, either direct or by contre-coup. Such effusions may be minute or considerable. Where they are minute, they occur mostly in a circumscribed form, more especially at the base of the brain, and more in the anterior and middle than in the posterior lobes. This is owing to the anatomical peculiarities of the inner surface of the skull, as at the base of it there are far greater irregularities in the shape of the bones than at the upper surface; and the anterior and middle lobes are more liable to be bruised on account of the numerous projections of bones with which they are in contact, while the posterior lobes are situated on a soft cushion, the tentorium cerebelli. Where these effusions are minute, the symptoms are those of encephalitis; while where they are large, the signs are those of cerebral hæmorrhage from other causes, as will be presently described.

Rupture of the capillary vessels of the brain also occurs in the course of that chronic inflammation of the grey matter which is found in some forms of insanity; and may be consequent upon embolism, and tumours which grow in the substance of the brain, and corrode the coats of the bloodvessels. It is also occasionally observed in leukæmia, where it is owing to accumulation of white corpuscles in the capillary vessels, which thus gradually become distended, blocked up, and ruptured.

As a rule, however, alterations in the quality of the blood, which were formerly believed to be important factors in the production of cerebral hæmorrhage, have only little influence in this respect. In scurvy and hæmophilia, for instance, a clot is rarely found, while ecchymosis, or infiltration of blood into the elements of the tissues, is more frequently observed.

3. In consequence of *contracted granular kidney*, which leads to blood-poisoning, and consequent overaction and hypertrophy of the coats of the arterioles, and hypertrophy of the left

ventricle. This connection was first pointed out by the late Dr. Kirkes, who traced, in a number of cases, kidney disease as the primary affection, and cerebral hæmorrhage as the last link in the chain of pathological events. *Thus intemperance will lead to apoplexy.* Alcohol, more particularly when taken in a concentrated form, as 'spirits and water,' and in habitually large quantities, gradually cauterises the glandular cells of the kidneys by which the drink has to be removed from the system. In proportion as these secreting cells are destroyed, the smallest renal arteries, which regulate the blood supply to the kidney, resist the entrance of blood, and become hypertrophied in their persistent endeavours to do so. Then as the surface of the kidney diminishes, the blood becomes poisoned with effete matters, a state which sets all the arteries throughout the system to contract in an excessive manner. In order to overcome the effect of this, the left ventricle increases in size and power, without there being any valvular disease to account for this. Dr. George Johnson explains these conditions by his theory of the 'stop-cock action' of the muscular coat of the arterioles. Long-continued over-action leads to hypertrophy of muscular tissue; and it is found that the inner longitudinal layer of fibres becomes equal in thickness to the outer circular layer. Thus, the onward movement of the blood is impeded; and the heart, resenting this resistance, has to act with increased vigour to overcome it, and carry on the circulation. Upon this, the arterioles contract more energetically still; and in this struggle between the two forces, the left ventricle becomes hypertrophied, and thus the pressure upon the arterial walls is increased. Thus there is considerable risk of hæmorrhage in those arterioles, the tissue of which does not become proportionally hypertrophied. In some cases it is found that the small subcutaneous arteries increase largely in size, while those of the brain do not do so in the same proportion; and wherever the excessive impelling force of the left ventricle is not exactly counterbalanced by corresponding hypertrophy of cerebral arterioles, an attack of cerebral hæmorrhage may be feared, simply from increase of vascular pressure. The heart under these circumstances reaches sometimes an enormous size, and

Dr. Broadbent [1] has recorded a case in which it weighed nineteen ounces.

Ordinary hypertrophy of the left ventricle from disease of the aortic valves does not lead to cerebral hæmorrhage. For many years heart disease use to be considered one of the chief causes of cerebral hæmorrhage, but it is probable that there has been confusion between rupture of cerebral bloodvessels, and embolism of cerebral arteries. The latter generally arises from previous endocarditis, and may occur with all the symptoms of apoplexy, rendering the diagnosis in some cases extremely difficult. *But heart disease by itself does not produce cerebral hæmorrhage.* Hypertrophy of the heart often acts only as a compensation for imperfect closing of the valves, and enables the circulation to be efficiently carried on, while without such hypertrophy circulation must be very much impeded. A large number of young and middle-aged persons are subject to various forms of heart disease, without becoming thereby liable to rupture of cerebral bloodvessels. Patients with emphysema and great dilatation of the right side of the heart, are often subject to giddiness, drowsiness, and other cerebral symptoms, owing to the brain being supplied with imperfectly aërated blood, and slow poisoning with carbonic acid; but cerebral hæmorrhage is quite exceptional in such cases, and occurs only when there is some peripheral obstacle to circulation, such as renal disease, which leads to increased tension in the bloodvessels of the brain.

4. By far the most important form of cerebral hæmorrhage, however, is that which occurs by rupture of the cerebral arterioles, after they have undergone the change known as *miliary aneurismal dilatation.* Miliary aneurisms are found in the large majority of cases of what is commonly called apoplexy, and constitute a disease of itself, which has special clinical and pathological features. These little aneurisms were first described by Virchow, but their pathological significance, and their constant relation to the common form of cerebral hæmorrhage, was only pointed out by Charcot and Bouchard, who called them 'miliary' from their resemblance to a millet seed. They are owing to a sclerous condition of the cerebral

[1] 'The Lancet,' Jan. 27, 1872.

arterioles, the middle coat of which is atrophied, while the connective tissue of the adventitia is hypertrophied. The vessel owes its power of resistance to the current of blood chiefly to the middle coat, and when the influence of this is removed, fusiform, cylindrical, or sacculated dilatations take place. The dilated adventitia adheres to the peri-vascular sheath; and various accidental causes which produce a temporary increase of pressure, may determine its rupture. These little aneurisms may, however, heal spontaneously by the formation of a clot in them, which then undergoes pigmentary degeneration.

Miliary aneurisms are slowly developed, and generally found to exist in considerable numbers. The cause of their formation is at present unknown, but it is probably a general one, for they have been discovered not only in the brain, but also in the œsophagus, on the visceral layer of the pericardium, the branches of the splenic artery, and the central artery of the retina. In the brain they are found in the fissures between the convolutions; in the corpus striatum and optic thalamus; in the white matter of the hemispheres, the cerebellum, and the pons Varolii. Their size varies from that of a millet grain to that of a large pin's head, and they are visible to the naked eye as dark red granulations. In my opinion the cause of the formation of miliary aneurisms has to be sought, first, in the tendency to vascular decay inherent to old age; second, in intemperance, combined with anxiety and hard work; and third, in hereditary predisposition.

When miliary aneurisms have become fully developed, comparatively slight causes may bring about their rupture. Thus we find that *cold* has a decided influence as an exciting cause of this form of cerebral hæmorrhage. It produces contraction of the bloodvessels of the skin, and thereby increases the tension in the cerebral arteries, while by stimulating the vaso-motor nerves, a dilatation of the cerebral arterioles is effected; under these circumstances a rupture of one or several miliary aneurisms is easily brought about. This explains why elderly persons are not unfrequently struck down with apoplexy in a cold bath, or immediately after taking one. Attacks of cerebral hæmorrhage are also more frequent during a severe winter than at other

seasons. Sudden changes in the temperature and the barometric pressure; staying in high elevations where the atmospheric pressure is feeble; and mental emotions which augment the activity of the cerebral circulation, also act as exciting causes of the attack. The same influence may be exercised by anything which increases the tension in the arterial system for a time, by impeding the return of the blood from the brain through the jugular vein. It is in this way that great muscular efforts, lifting heavy loads, violent coughing, sneezing, or vomiting, seem to bring on a stroke. Thrombosis of the sinuses of the dura mater, compression of the jugular veins or the superior cava by tumours, and finally lesions of the right heart, and the pulmonary artery, may lead to a similar result.

In this place I must say a few words about the so-called *apoplectic habit.* Where the body is stout, the neck short, the shoulders broad, the chest well or powerfully developed, the abdomen large, the flesh abundant, and there is general and chiefly abdominal plethora, both doctor and patient used to feel great apprehension of an approaching attack of paralysis. But the influence of the full habit of body has been very much exaggerated; it certainly predisposes to attacks of cerebral congestion, but not of hæmorrhage, to which latter lean and anæmic people are quite as much exposed as those of an opposite habit.

The quantity of blood which escapes in consequence of the rupture of one or several miliary aneurisms, varies from a few drops to about sixteen ounces. This effused blood tears up the brain-tissue, and produces a cavity which may occupy the better portion of an entire hemisphere. There is generally only one clot, which occupies some portions of the grey matter. Most cases of cerebral hæmorrhage result from rupture of those arterioles which arise from the mid-cerebral artery, and proceed through the substantia perforata lateralis to the corpus striatum. The blood penetrates to the centre of Vieussens, pushes the thalamus opticus inwards, and raises and finally ruptures the corpus striatum. It will then invade the lateral ventricles, and through Monro's foramen, break into the third; it may also break through the island of Reil, and following the fissure of Sylvius, fill up the fourth ventricle, or tear up the lower

portion of the infundibulum, and spread to the base of the brain.

Next in frequency to the corpus striatum comes the thalamus opticus. Andral has collected the autopsies of 392 cases, in 202 of which both corpus striatum and thalamus opticus were simultaneously affected; in 61 the corpus striatum alone, and in 35 the optic thalamus alone: in 27 that portion of the hemisphere which is above the centrum ovale of Vieussens; in 16 the lateral lobes of the cerebellum; in 10 that portion of the brain which is in front of the corpus striatum; in 9 the corpus callosum; in 7 the posterior lobes of the brain; in 5 the middle lobes; in 3 the peduncles of the brain; in 1 the crus cerebelli; in 1 the corpora olivaria, and in 1 the petuitary body. In the majority of cases there is only one clot, which, however, may be owing to the rupture of a number of small aneurisms; but Dr. J. Ogle has recorded a case in which separate clots were discovered in the right corpus striatum, the left thalamus opticus, and the pons Varolii. There is, indeed, no portion of the brain which may not become subject to hæmorrhage.

After death the convolutions of the suffering hemisphere are found flattened and anæmic, from pressure of the clot upon the cerebral arterioles which thereby become empty; the veins of the pia mater are also empty. The effused blood coagulates rapidly; it looks like currant jelly, and is mixed with débris of the cerebral matter, which are plainly seen when a jet of water is made to play upon the parts and washes the blood away. The parts surrounding the clot are softened. Where the patient does not succumb to the stroke, a necro-biotic process of absorption sets in after the hæmorrhage is arrested. The blood-corpuscles and nerve-fibres undergo fatty degeneration; the serum is absorbed; granular bodies are formed out of the débris; and pigmentary granulations and crystals of hæmatine, hæmatoidine, and amorphous granulations of hæmatosine, form a semi-liquid mass, which is at first black, and afterwards assumes an ochrous appearance.

At the same time a subacute inflammation takes place in the neuroglia, or cementing connective tissue of the brain-matter, which is in many cases of a healing kind, but occa-

sionally assumes a hyper-acute character, and runs a short and fatal course. The vacuum which is caused by the contraction of the clot, is filled up by the effusion of a liquid, which is at first homogeneous, but gradually assumes the characters of connective tissue, which is deposited in concentric layers, and ultimately forms a capsule, which encloses the remains of the clot. This limiting membrane secretes serum, which, by imbibing the decayed matters of the cavity, facilitates their absorption. The liquid is either colourless or has a yellowish tinge. The size of the cyst varies from that of a pea or cherrystone to that of a small apple; but sometimes the inflammation extends to a considerable distance from the original lesion, and leads to sclerosis. When the clot has been large, a considerable loss of substance must be the result; the membranes are depressed, and only separated from the ventricles by a thin layer of tissue. As time goes on the cyst-wall becomes hardened, and completely separates anything yet remaining of the clot from the surrounding cerebral matter in which it lies imbedded.

Apoplectic cysts are not found in the cortical substance, but only in the interior of the hemispheres, which is owing to the circumstance that the vacuum which is caused by hæmorrhage in the cortical substance is filled up by the effusion of a corresponding quantity of meningeal serum, while in the hemispheres it is filled up by the cystic liquid just described. In the cerebral convolutions where such an effusion has taken place, an accumulation of yellow or brownish pigment, which is intimately connected with the pia mater, is sometimes the only remnant of the clot.

These changes vary considerably, according to the constitutional powers of the patient; and a small clot in an aged person with enfeebled systemic energy, where the healing process is slow, may cause more loss of function than a clot considerably larger in a comparatively young man of otherwise unimpaired vigour. In old, decrepit, exhausted, and badly-nourished persons, there is less contraction of the clot, and more liability to inflammatory irritation with its well-known consequences. In some cases, a subacute inflammation goes on for a long time in the neighbourhood of the original lesion,

and this is indicated by rigidity and contraction of the paralysed muscles, with spasms and general irritability, which is sooner or later followed by exhaustion and collapse.

The absorption of the clot generally commences in about four to eight days after the seizure; the formation of the cyst commences about three weeks afterwards, and is generally finished in three months. If much liquid accumulates in the cyst, a great amount of wasting may take place in the cerebral matter, and which in no way differs from ordinary senile atrophy. It is not at all proportionate to the size of the original effusion, but may be very extensive where the clot has been very small.

Cerebral hæmorrhage never kills with the same rapidity as some forms of heart disease; and what French authors call 'apoplexie foudroyante' does not really deserve to be compared with the rapidity of a lightning stroke, for even in those cases which are most rapidly fatal—viz., where the bleeding takes place from the rupture of an ordinary aneurism of one of the basilar arteries, the least interval between the commencement of the symptoms and the fatal issue has been seven minutes, and it will commonly take from fifteen to thirty minutes. Syncope, indeed, may kill as quickly as lightning, but the term 'apoplexie foudroyante' is a misnomer.

In the large majority of fatal cases from six to twelve hours elapse between the beginning of the illness and its end; and that this should be so is easily accounted for by the circumstance that the size of the miliary aneurisms is too small to allow the rapid escape of a quantity of blood sufficient to kill. It is well known that the brain is able to bear the presence of a small quantity of blood in some of its parts without resenting it violently; and the anæmia of the organ which is caused by the hæmorrhage, and the shock to the brain which accompanies the effusion, must have reached a considerable degree before they prove to be incompatible with life.

Premonitory Symptoms.

Some observers have denied that cerebral hæmorrhage is preceded by any symptoms showing that the brain is becoming

liable to an attack of apoplexy; and there can be no doubt that a number of symptoms which were previously put down as such are in reality owing to incipient softening of the brain. These are loss of memory, embarrassed speech, inability to write or spell correctly, loss of the capability of carrying out certain finer movements, such as threading a needle, buttoning a shirt-sleeve, etc., and impaired intellect. There are, however, other signs which plainly belong to the disease which we are now considering, and which cannot be reasoned away by any specious argumentation; although it is quite true that occasionally the patient is struck down in apparently excellent health.

An important premonitory symptom is bleeding at the nose, which, unimportant in children, becomes of grave significance in elderly persons. It may precede an attack of apoplexy by a few months, or even several years. Mydriasis and paralysis of the rectus externus muscle likewise occur, and are then probably owing to minute bleeding in the neighbourhood of the nuclei or roots of the third and sixth nerve.

Apoplexy of the retina occurs from various causes, such as impeded general circulation from disease of the heart, liver, kidneys, and womb; or where there is an obstacle to local circulation, as when a tumour presses upon the optic nerve, either in the orbit or within the skull. It is, however, frequently connected with general vascular decay, and may, if recognised by an examination with the ophthalmoscope, lead us to suspect similar changes in the cerebral bloodvessels. Sometimes the hæmorrhage occurs just in the yellow spot, and the patient then becomes blind in a few minutes; while where the effusion takes place nearer the periphery, and is not very large, the sight may be very little affected. The ophthalmoscope in such cases, however, always shows that we have to do with a grave lesion, which, even if the other symptoms are slight, will probably sooner or later cause incurable blindness; for the disease is extremely liable to relapses, and repeated effusions of blood must eventually lead to atrophy of the optic nerve and retina.

Apoplexy of the retina generally occurs in advanced life, that is, after fifty years of age, and has been more frequently seen in men than in women. It is probably always, sooner or

later, followed by cerebral hæmorrhage. Dr. Berthold[1] has recorded the case of a woman aged 40, who came to consult him for well-marked hæmorrhage into the macula lutea, and who, while under examination, was seized with an attack of cerebral hæmorrhage which proved fatal.

The ophthalmoscopic appearances vary somewhat in cases of this class. There is either a number of small round circumscribed spots of blood; or there are longitudinal streaks or brush-like bands, when the blood has been effused within the nerve-fibre layer; or there are large irregular patches in the neighbourhood of the veins, which are dark and greatly dilated and tortuous, while the arteries appear small and anæmic. The disc is blurred and has an indistinct outline, or it appears very dark from hyperæmia of the veins. The effusion has more tendency to spread into the choroid than into the vitreous body, but if it breaks into the latter it causes dense opacities. The blood ultimately undergoes pigmentary degeneration.

Other symptoms which I have known to precede an attack of cerebral hæmorrhage, are a feeling of malaise about the head, which is chiefly experienced on rising in the morning, but goes off as the day advances. Emotions, annoyance, and indigestion are apt to bring it on again at any time of the day. Drowsiness, which can be shaken off by an effort, and disinclination to work, are likewise experienced. Giddiness on stooping, or on quickly turning the head; severe paroxysmal headache; attacks of glimmering, so that the patient sees stars, sparks, and other luminous appearances in a vibratory or wave-like motion; neuralgic pains in the sphere of the brachial plexus, sometimes singling out the little finger with particular vehemence; flushings of the face, with hot ears and cold hands and feet—all these symptoms may come on separately or conjointly, and point to impaired cerebral circulation from sclerosis of the arterioles, being due to anæmia rather than to hyperæmia.

The attack of cerebral hæmorrhage generally commences with giddiness, specks before the eyes, noises in the head, and tingling in the limbs; the patient speaks thickly, staggers, falls, and suddenly loses his consciousness; if taken up, he falls back, an inert mass. He is now insensible to stimulation,

[1] 'Klinische Monatsblätter für Augenheilkunde,' April, 1874.

while the automatic movements of respiration and the heart's action continue. This is the ordinary sequence of events in corpus striatum and thalamus opticus hæmorrhage. Where the blood breaks into the meninges or the lateral ventricles, the limbs of one side of the body are contracted and convulsed; while if there is hemiplegia without loss of consciousness, the hæmorrhage has most probably taken place into the cerebellum.

It is not easy to account for the *loss of consciousness* where the effusion in the central ganglia is small. This cannot be attributed to 'congestion' as was formerly done; for there is no congestion. Nor is the anæmia of the brain produced by the bleeding sufficient to account for the coma, as in some cases the effusion is very small, and nevertheless causes insensibility. It is also well known that cerebral anæmia does not invariably lead to unconsciousness. There is, for instance, great cerebral anæmia in cholera, but no unconsciousness; and on the other hand, in cysts and abscess of the brain, a great deal of pressure may exist without producing insensibility. Much, no doubt, depends upon the suddenness with which the symptoms are produced: and a rapidly-occurring injury is more likely to cause unconsciousness than one which takes place so slowly as to allow the brain to become accustomed to the pressure. A patient may live for a considerable time with cerebral abscess, and then suddenly die comatose, when the abscess bursts, and the lateral ventricles become flooded with pus. In such a case there is no actual increase of intracranial pressure, but merely a transfer of pus from one portion of the brain to another, which latter is less able to bear it than the former. Pagenstecher's experiments on dogs have shown that an injection of about twenty-three fluid ounces into the cavity of the skull is required to produce symptoms of compression; and the average weight of the clot is from four to six ounces only.

Apart from the slowness or suddenness with which the pressure is produced, we find that when certain portions of the brain are affected by hæmorrhage, loss of consciousness is more apt to come on than when others suffer; and it is chiefly the central ganglia which resent the irruption of blood in this manner. We are therefore obliged, for want of a better explanation, to assume *shock of the brain* by the sudden invasion of

blood into the structure of the central ganglia, in order to account for the loss of consciousness. This shock is transmitted from the corpus striatum or thalamus opticus to the corresponding hemisphere, and from there, by the numerous commissural fibres which connect the two halves of the brain, to the opposite hemisphere. It may even be propagated to the spinal cord, which is shown by the abolition of reflex movements. In most cases, however, reflex excitability is increased, by removal of the inhibitory action exerted by the brain (p. 41); and if the sole be tickled, the leg is wildly thrown about, or both legs or the whole body respond to the stimulation.

In the further progress of cerebral hæmorrhage we find that the features become vacant and devoid of all expression. The face is distorted, and either livid and swollen or ghastly pale. The cheeks are flabby and blown out by each expiration. The mouth is either uniformly open, or half open, and drawn to the opposite side; at each expiration the commissure of the lips is raised at the paralysed side, and lets a frothy saliva run down the chin. The naso-labial sulcus is effaced, or appears drooping and drawn to the non-paralysed side.

The speech is entirely lost. A strong light, loud sounds, powerfully smelling or sapid substances, no longer convey any impressions to the brain; the patient cannot be roused by shouting to him, or by any other of the usual modes of reviving consciousness. The conjunctiva is insensible, either in both eyes or only in the eye of that side which is afterwards found paralysed. The pupil of the paralysed side is generally found dilated; but this is by no means invariable, as the iris receives nervous supply from various sources, and there may be constriction or dilatation, or inequalities in the size of both pupils, according to irritation or paralysis of certain portions of the nervous system of the iris. Irritation of the third nerve causes constriction of the pupil; but when the attack is accompanied by severe convulsive seizures, the pupils become extremely wide, and contract after the convulsion is over. If both pupils are extremely contracted, the hæmorrhage is generally in the pons Varolii, and is sometimes confounded with opium-poisoning.

The superficial veins of the head and extremities are either gorged with blood or scarcely visible. The skin may be quite

dry, or bathed in clammy perspiration. Where from imperfect respiration the blood does not receive a full supply of oxygen, the skin generally appears livid. These symptoms also depend to a great extent upon the previous habit of the patient. In the full-blooded and those liable to congestions of various parts, we find gorged peripheral circulation; while in anæmic persons the skin is pale and cool.

A severe attack of cerebral hæmorrhage is always accompanied with involuntary evacuation of the urine and fæces. The sphincter ani does no longer offer any resistance to the introduction of a finger or an instrument. The automatic movements of circulation and respiration, which at first continued much in the same manner as they do during a heavy sleep, after a time become affected in their turn. Inspiration is short, superficial and irregular; the soft palate is heard to flap to and fro, producing stertor; and from having been accelerated, respiration becomes retarded and intermittent. Mucus accumulates in the air-passages, and laryngeal, tracheal, and bronchial râles are heard. A frothy liquid, which is a mixture of saliva, mucus and air, is seen to run down the chin. The pulse is large, hard, and incompressible, more especially in the carotids—not from congestion, as has been erroneously supposed, but from the resistance encountered by the current of the blood, which cannot enter the intracranial bloodvessels, compressed as they are by the effusion. This symptom, however, loses its significance if there is hypertrophy of the left ventricle, and equally strong pulsation of other arteries.

For some time during the progress of apoplexy, the symptoms are, as it were, of a mixed character, viz. partly paralytic and partly spasmodic. The former, which have just been described, are owing to the pressure of the escaped blood upon the brain matter, while the latter are caused by the irritation of the brain from the blood undermining the central ganglia, previously to its destroying their texture.

While the corpus striatum is being irritated and destroyed by the blood flowing out from the opening of one or several miliary aneurisms, there are often severe convulsive attacks shaking the arm and leg of the opposite side of the body; or there is great rigidity of the muscles of the limbs.

An important and pathologically interesting symptom is the *conjoint or synergic lateral deviation of the head and eyes*, which is not unfrequently observed in corpus striatum hæmorrhage, and also occurs where blood is effused into the meninges. The head and eyes look away from the paralysed side, and are turned towards that side in which the cerebral lesion has occurred. Thus, in right hemiplegia and aphasia from a clot in the left hemisphere, the head and eyes are turned to the left side. This is sometimes seen shortly after the commencement of the attack, when the patient is quite unconscious; but it may persist after the return of consciousness, and it is then seen that the patient has no power in overcoming the deviation. Sometimes it is so complete that the irides almost disappear; while at other times it is less marked. When such is the case, the patient may, at our request, turn the eyes still further away from the paralysed side, but not in the opposite direction; but sometimes he is able, on making an effort, to turn the head in the opposite direction.

Where this deviation is after a time followed by deviation to the opposite side, we may conclude that the effusion has entered the lateral ventricles, or that a new cavity is forming in the other hemisphere. This symptom, which is purely spasmodic, and not paralytic, is also occasionally seen in embolism of the mid-cerebral artery, and after a succession of epileptic seizures; and it always denotes a sudden and extensive injury to the brain, which mostly proves fatal.

Conjoint deviation of the head and eyes is produced by irritation of the following cerebral nerves: 1st. the sixth, which causes external strabismus on the side of the lesion; 2nd. the third nerve of the opposite side, which causes internal strabismus of the opposite side; and 3rd. the spinal accessory nerve, which produces rigidity of the sterno-mastoid muscle, and deviation of the head.

The *temperature* of the body undergoes at the same time considerable alterations; for while in the commencement of the attack, and particularly where the hæmorrhage is not abundant, the body-heat remains normal, after ten or fifteen minutes the thermometer placed in the rectum or armpit, shows signs of falling, and may go down to 95°. This fall must be looked

upon as a symptom of irritation or spasm of the controlling centres of heat-production, and has the same clinical significance as convulsions of the muscles of the extremities.

After a time, which varies from thirty minutes to thirty-six hours, the spasm in the centre ceases, from the blood breaking into the lateral ventricles and into the fourth. The symptoms then become purely paralytic in their character; the lateral deviation of the head and eyes disappears; convulsions and rigidity cease, and the body-heat begins to rise more or less rapidly. Where this rise is extensive, it has the same unfavourable meaning as a great fall in the commencement; and the mercury, now at 103° to 105°, corresponds in significance to the low readings of the first period. More especially when the temperature runs up rapidly, it is a sure sign of collapse, which is soon followed by death.

On the other hand, we find cases in which the thermometer, after a comparatively slight fall, rises only little, if at all, above the normal mean, but remains stationary at 98° or 99°. This means that the hæmorrhage has been arrested; the opening in the miliary aneurism having been as it were sealed up by contraction of the clot, before the blood could have broken into the lateral ventricles. There is therefore now only a comparatively small cavity filled with blood, which may be expected, in course of time, to undergo its appointed changes. There is no lateral deviation of the head and eyes; no rigidity or convulsion of the limbs; the body does not appear so completely relaxed as before; the difference between the sound and the paralysed side becomes more marked, one being quite relaxed, while the other offers some resistance on being moved, or carries out semi-voluntary movements; the patient can be roused on being spoken to; he begins to talk in a dreamy fashion, and the coma gradually lightens into consciousness. The pulse becomes steadier, and the respiration more regular, although the contraction of that side of the diaphragm which corresponds to the paralysed side, is more feeble than that of the other side.

The patient has now escaped death from collapse, but he is still liable to be carried off by the consecutive cerebral fever from secondary encephalitis, which in cases that are to end

fatally, is apt to come on after the partial recovery just described has taken place. The first symptoms of cerebral fever are generally headache, restlessness, and sensory delusions; the patient is throwing himself about in a terrified manner; there is moaning, and muttering delirium; but the pathognomic symptom in such cases is an acute bedsore on the buttock of the paralysed side (*tâche cérébrale*).

An erythematous spot, or macula, appearing on the second, third or fourth day, on the part just mentioned, almost invariably heralds a fatal termination of the case; for this change in the nutrition of the skin and subjacent parts shows paralysis of the intracranial trophic centres, which precedes paralysis of the volitional centre only by a short time. The pathology of the process is the same as that of ulceration of the cornea after section of the fifth nerve. The cerebral macula has an irregular shape and varies in size; from being pink at first, it gradually changes into purple. Pressure will cause it to disappear, showing that in the beginning there is only hyperæmia. Rapidity of progress, however, a hyper-acute course, is characteristic of the affection. The hyperæmia is soon followed by effusion of serum, and phlyctænæ are formed, which contain a liquid that is at first colourless, but shortly becomes sanguinolent and livid. The raised cuticle then gives way, and an open sore is left, which has a scarlet surface, and appears covered with livid granulations. Gangrene is now fully established, and if the patient survives long enough, inflammation sets in, by which the gangrenous parts are to be eliminated.

It is quite evident that pressure alone is not sufficient to account for the appearance of this acute decubitus; for the pressure is equal for both buttocks, and yet there is no eschar formed on the non-paralysed side. Moreover, it is not prevented by letting the patient rest habitually on the non-paralysed side, so as to avoid pressure on the paralysed side. Nor can the irritating effect of the urine on the skin be accused; for Charcot, who has studied this condition more particularly, has found that it occurs also where the urine is drawn off with the catheter at short intervals by day and night, so as to avoid such an influence. It is indeed quite different from chronic decubitus which occurs habitually, in consequence of prolonged

pressure on the back and other parts, in the course of protracted disease, where the patients are bedridden, and where there is frequent involuntary evacuation of the urine and fæces, which aids in the production of gangrene. Moreover, the bedsore which occurs in protracted cases of spinal and other diseases, affects more the sacrum than the buttock. Acute cerebral decubitus must therefore be ascribed to collapse of the trophic centres in the brain. A similar occurrence takes place in severe cases of myelitis, where a decided difference is found to exist between acute decubitus from collapse, and chronic bedsores from pressure. (*Vide* Chapter V.)

In some cases appearances similar to those just described are observed on the heel, ankle, and knee of the paralysed leg; and where the effusion has burrowed its way into the lateral ventricles, phlyctænae may appear on both nates.

The viscera sometimes show traces of similar affections. Congestive patches or real ecchymoses are sometimes seen at post-mortem examinations in the pleura, pericardium, and the mucous membrane of the stomach, and which are undoubtedly produced by the same cause.

A rise in the temperature may be expected with certainty after the cerebral macula has begun to form; and the mercury will in a comparatively short time run up to 103°, 104°, and even 105°. At the same time, other symptoms manifest themselves which point to the impending dissolution. The patient throws himself about in a restless manner, and a low muttering delirium sets in, which is occasionally broken by loud moanings. The pulse becomes small and compressible, and runs up to 120 or more beats. Respiration is hurried and superficial, at the rate of 42 to 60 in the minute; and the abdomen is drawn in during inspiration, showing commencing paralysis of the phrenic nerve and the medulla oblongata. The extremities become cyanotic, and completely relaxed. The face, and sometimes the whole body, is bathed in clammy perspiration; the neck is flabby, and the head will retain any position in which it is placed. Pulmonary complications sometimes set in before death, and the temperature occasionally reaches its maximum shortly after the fatal issue.

Cerebral fever rarely occurs later than the fourth day of

the attack; and if the patient, therefore, survive the fifth or six day without there being phlyctænae and undue elevation of temperature, his prospects of recovery become favourable. Nevertheless the condition of his arterial system renders further attacks probable.

When the attack and the reaction are over, that is, in about ten or eleven days after the commencement of the illness, we have simply to do with symptoms owing to the presence of the clot. We now find either complete hemiplegia, i.e. paralysis of one side of the body; or it is seen that a gradual recovery of voluntary power takes place, and which is in direct proportion to the anatomical changes which have been previously described (p. 95).

Motor Paralysis is the most striking consequence of an ordinary attack of cerebral hæmorrhage, and its degree depends upon the quantity of the effusion, and the part of the brain which has suffered from it. Where the entire corpus striatum is destroyed, there is complete and permanent hemiplegia of the opposite side of the body. While, however, the commands of the intellect can no longer be carried out by the paralysed muscles, the reflectory and emotional movements do not suffer, and may even be increased. The patient may be unable to laugh at the affected side when told to do so; but when his emotions are excited he may laugh on both sides of the face. Tickling of the sole causes extensive movements of the paralysed leg, or both legs, and on gaping, arm and leg are drawn upwards; showing that the spinal cord still retains its influence over the paralysed muscles, while the inhibitory influence of the brain is withdrawn. At a later stage of the complaint, however, there is generally diminution, and sometimes even utter abolition, of reflex movements.

I have already adverted (p. 48) to the theory of Dr. Broadbent, according to which the parts which are really paralysed are those which have the power of acting singly, and independently of the corresponding parts of the opposite side; while those muscles which escape act laterally, or at least in concert with the corresponding muscles of the opposite side; and I shall now proceed to consider the paralysis of motion after corpus striatum hæmorrhage, in accordance with this theory, which is fully borne out by the facts of the case.

The muscles which escape are chiefly those of the eye, neck, back and chest. The movement of the eyes, which are under the influence of the third, fourth, and sixth nerves, are generally perfect; the patients may also move their head forwards, backwards and sideways; the motions of the chest do not differ on either side, and inspiration and expiration is not only performed automatically, but may also be carried out by the command of the will. The paralysis, however, is marked in the face, arm, and leg of the side which is opposite to the lesion.

The paralysis of the face is always incomplete, and thereby easily distinguished from facial palsy caused by disease of the portio dura. In the latter affection every muscle animated by that nerve has lost its motive power; and the paralysis appears particularly striking in the orbiculares palpebrarum et oris, owing to which the patient cannot close the eye of the affected side, and has lost the power to purse up his lips, to kiss and to whistle. In cerebral palsy, on the other hand, the patient can always close his eye, and has lost only very little power over the orbicularis oris, being able to blow and to whistle. The muscles which chiefly suffer in this form of facial palsy are the straight muscles going to the lips and the angle of the mouth; that is, the levator anguli oris, labii superioris et alæ nasi, and the zygomatics. The affected lip therefore droops and is drawn to the healthy side, and this deformity augments when the patient speaks or laughs. The cheek is flabby, and does not partake in movements of the face: while the lip of the opposite side stands higher, being drawn upwards and outwards. The nostril is narrowed, and during expiration the cheek is puffed out like a sail by the wind, there being no muscular tone to resist the passage of the air. We also find that, while in paralysis of the portio dura the orbicularis palpebrarum is so completely paralysed that the patient cannot shut his eye even if he makes the greatest efforts to do so, and the eye remains open even during sleep, the patient affected with cerebral facial palsy can shut it apparently quite well. Yet the muscle is weakened, for the patient cannot squeeze the eyelid as tightly over the eye as he can do with the other; when the eyes are closed, that of the paralysed side is less so than the other; and while he can close the eye of the unaffected side by itself

unilaterally, he cannot close that of the affected side by itself, or at least experiences considerable difficulty in doing so.

The corrugator supercilii and the occipito-frontal muscles are likewise weakened; for although the patient can frown a little when told to do so, yet there is a perceptible difference between the two sides. Moreover, habitual want of tone is shown by the diminution of the horizontal and perpendicular wrinkles on the forehead. In facial palsy from affection of the portio dura these wrinkles disappear completely on one side; in cerebral facial palsy they do not disappear, but are less marked.

The minor portion of the fifth nerve is likewise affected; and if the patient be requested to close the jaws firmly, it is found that the temporal and masseter muscles of the sound side contract sooner and more energetically than those of the paralysed side. Mastication is therefore not so easy, and the patient cannot well move the bolus in the mouth, owing to partial paralysis of the buccinatorius and the tongue.

The tongue is in the large majority of cases deviated towards the paralysed side. This apparently strange fact finds its explanation in the peculiar action of the genio-glossus muscle, which draws the tip of the tongue, when the organ is protruded, in an oblique direction towards the opposite side. In health both genio-glossi act together, so that when the tongue is protruded, the lateral movements are neutralised by the antagonistic action of the two muscles. But as soon as one genio-glossus is paralysed, the other loses its antagonist, and directs the tip of the tongue to the opposite, that is, the paralysed side. In a few cases we find indeed that the tip of the tongue is directed to the healthy side; but these are instances, not of corpus striatum hæmorrhage, but of lesions of the medulla, where there is crossed paralysis of the tongue and the limbs.

While therefore the unilateral action of the tongue is interfered with, combined movements may still be carried out by it. Thus its two lateral halves appear generally symmetrical, and it may be flattened and elongated as a whole.

The muscles of the back almost always act together; and even when the body is inclined to one side, the muscles of one

side produce, and those of the other side regulate, the movement, in order to prevent falling over. The same is the case with the muscles of the neck.

The diaphragm is not affected, as the phrenic nerves continue to act normally; but the recti abdominis muscles are weakened on the paralysed side. This is seen when the patient attempts to raise himself from the recumbent to the sitting posture by the help of these muscles. If we then place the hand on the abdomen, we feel that the muscles of the paralysed side do not contract so quickly nor so energetically as those of the healthy side.

The greatest degree of paralysis is found in the *arm*, which acts habitually more singly than any other portion of the body, and if recovery takes place, this is in the direction from the centre to the periphery. In severe cases the shoulder-blade is quite devoid of motion, from paralysis of the serratus, trapezius, latissimus dorsi, and other muscles. The arm itself cannot be abducted, from paralysis of the supra-spinatus and deltoid muscles, nor adducted, through paralysis of the coraco-brachial; it cannot be drawn forward, as the subscapularis and the pectoralis major muscles do not obey; nor backwards, from want of power in the infraspinatus and latissimus. Flexion and extension, supination and pronation are likewise impossible, through paralysis of the muscles intrusted with those movements.

Where no or only slight improvement takes place in the muscles which move the arm and forearm, contractions of their tissue are not slow to appear. The flexor muscles are generally much more contracted than the extensors. The arm often appears adducted to the side, from contraction of the coraco-brachial muscle, and the forearm is bent on the arm, from contraction of the biceps. The triceps being an extensor, is more paralysed than contracted; but it does not altogether escape contraction, which is shown by its becoming so far rigid as to prevent complete passive flexion of the elbow, so that the tips of the fingers cannot be placed on the acromion of the same side. Endeavours to carry out these movements cause pain.

The wrist is particularly affected. The four typical movements of it are dorsal and volar flexion, and radial and ulnar

flexion, each movement being performed by two muscles, viz., volar flexion by the flexor carpi radialis and ulnaris; dorsal flexion (commonly called extension) by the extensor carpi radialis and ulnaris; radial flexion, by the flexor and extensor carpi radialis, and ulnar flexion by the flexor and extensor carpi ulnaris. Amongst these four movements, that of dorsal flexion is most obstinately and permanently affected in cases of hemiplegia; and a patient who has recovered this action may be considered fortunate. The contractions are most severe in the muscles which provide for volar flexion; and the flexor carpi radialis suffers more than the flexor carpi ulnaris.

Like the wrist, the *fingers* have four different kinds of motion, viz. flexion, extension, abduction and adduction, which are produced by four separate muscles for each finger, and which all suffer more or less in hemiplegia from corpus striatum hæmorrhage, either by contraction or paralysis. Among these muscles, the extensors and abductors suffer chiefly from paralysis, and the flexors and abductors from contraction. Moreover the thumb and the little finger lose a movement peculiar to them which is carried out by two separate muscles, viz. that of opposition of their volar surfaces. The contraction of the flexors is often so strong that the fingers are forcibly bent into the palm, and that the growing nails irritate the skin. The adductor and opponent of the thumb are in such a state of contraction that the last phalange of the thumb touches the second or even third finger with its volar surface; and the movements of abduction or extension are only rarely recovered from.

Although at first the *leg* is quite as much paralysed as the arm, we find that in most cases a difference soon becomes apparent between the upper and lower extremity; for while the arm remains unable to obey the orders of volition, the leg begins to execute sluggish movements, and the patient is sometimes able, within the first week of the stroke, to get out of bed and to hobble about the room, with the arm dangling by his side like a piece of inanimate matter. The same law which obtains for the upper extremity, also holds good for the lower, viz. that recovery progresses from the centre to the periphery. The hip-joint therefore gains its motion before the knee, and the latter before the foot and the toes. An occasional

exception to this rule is made by the toes, which in some instances recover before the ankle-joint. In these cases great care must be taken to distinguish voluntary from reflex movements, which latter are sometimes excited by the least cause, and which are apt to deceive the observer into the belief that there is recovery of volitional power. In the lower extremity likewise the extensors are chiefly subject to paralysis, and the flexors to contraction. In most cases the latter do not recover thoroughly, in consequence of which the paralysed leg appears shorter than the opposite one, and a limp in the gait is the consequence. In some cases the patient walks chiefly with the muscles of the back and hip-joint, those of the thigh, leg and foot remaining more or less inactive. This explains the peculiar swinging motion of the leg which is so often seen in hemiplegia from corpus striatum hæmorrhage.

The sphincter muscles generally recover completely within a few days of the stroke, unless the patients are very much advanced in age, or the effusion of blood has been very copious.

Combined movements which require a certain amount of skill, especially where the right hand is concerned, such as writing, sewing, playing on musical instruments, dressing, and more particularly buttoning, continue awkward and troublesome long after a fair share of coarse muscular power has returned.

Associated movements may occur together with involuntary or reflex movements. Thus the paralysed limbs are seen to jump about during the acts of gaping, sneezing, coughing, and laughing. Voluntary movements of the healthy limbs of the opposite side have no such effect. The muscles of the face sometimes recover their physiognomical expression for a short time, when the patient is under the influence of excitement or indignation, and lose it again as soon 'as he has calmed down.'

Aphasia, or loss of language, being more frequently produced by softening than by hæmorrhage, will be considered in the following chapter.

The automatic movements of circulation and respiration are generally impaired for some time after the seizure. The pulse is often irregular and intermittent. Respiration is sometimes entirely diaphragmatic. There is difficulty in swallowing; regurgitation of liquids is not uncommon; digestion is tardy, and the action of the bowels sluggish.

Do patients ever completely recover from an attack of corpus striatum hæmorrhage?

This question used formerly to be unhesitatingly answered in the affirmative. Even so recent an observer as Durand-Fardel states that of twenty-seven such cases which came under his care, nine were cured, two nearly so, four remained weak in the affected limbs, one retained difficult articulation, one became imbecile, and ten remained hemiplegic. This proportion of recoveries, however, does not really occur; and if we were to accept it without reserve, it would imply a far greater vitality of the previous than the present generation, or more effective modes of treatment employed by our predecessors in practice than by ourselves. We shall be nearer the truth in assuming that our search for symptoms of disease is now keener than previously, and that we consequently discover them more frequently. Having had more than four hundred cases of hemiplegia from corpus striatum hæmorrhage under my care in hospital and private practice, I am enabled to state that complete recovery is the exception, and that even in those patients who professed to enjoy good health after a seizure, the memory was as a rule less ready, the speech less fluent, the power of application less enduring, and the sense of touch and the co-ordination of movements less quick than before the attack.

Nor should it be a matter of surprise that such patients do not as a rule entirely regain their faculties. The blood which has been effused, must in the nature of things destroy a number of nerve-cells which can never be regenerated, and whose place will ultimately be taken by connective tissue. The eventual loss of function will be proportionate to the extent and importance of the destroyed parts, and to the degree to which their place can be taken by allied structures in the neighbourhood of the lesion. A somewhat analogous case is that of a man who has had a portion of his lung consolidated by tubercular deposit, and who, although the activity of the disease may have been arrested, and retrogressive changes taken place in the tubercle which renders it innocuous to the system, can never be so strong as he might have been with the entire organ in full functional activity, although he may certainly enjoy a measure of health and strength.

The extent to which recovery of function may take place depends—

1. Upon the quantity of blood which has been effused. This varies from a few drops to several ounces, and the less escapes, the better will be the patient's prospects.

2. Upon the portion of the brain in which the effusion has taken place. Hemiplegia affecting the right side of the body is not only more serious in its aspect than the left, because it is almost invariably combined with loss or great impairment of speech, which is rarely entirely recovered, but it also appears that the patients regain the use of the left side more readily than that of the right side of the body; to which must be added the circumstance that the left arm and hand are not nearly so essential to the patient as the corresponding limbs of the right side.

3. Upon the manner in which the effused blood is disposed of (p. 94); and lastly

4. Upon certain secondary alterations which in many cases occur subsequently in the nervous centres, peripheral nerves and muscles, and which begin probably soon after the stroke. If any such occur, ultimate recovery is rendered impossible. The chief symptom in these cases is that the paralysed limbs become permanently contracted. Some observers have thought this to be owing to secondary encephalitis, but the researches of Türck, Charcot, and Vulpian have shown that the nature of the affection is *sclerosis of the lateral columns of the spinal cord.* This sclerosis proceeds in a descending direction from the seat of the lesion, affecting the cerebellum, the peduncles, the medulla oblongata, and the spinal cord. It is chiefly seen after corpus striatum, and less after thalamus opticus, hæmorrhage. A grey band is found on the cerebral peduncle corresponding to the destroyed corpus striatum, occupying the external, middle, or internal portion, according as the primary lesion has affected the external, middle, or internal part of the striated body. This grey band may be traced throughout the medulla and the lateral column of the cord. The medulla is flattened and lessened in size, and the pyramid small and grey. The degenerated fasciculus crosses over in the anterior pyramid to the opposite side, and

the lesion in the cord is therefore found in the side opposite to the cerebral lesion. The sclerosed part is situated just before the posterior roots, in the lateral columns, and quite limited in extent. To the naked eye these changes are visible about four or six months after the stroke, but with the aid of the microscope they may be discerned much sooner. There is a formation of granular cells, and the nerve-fibres and coats of blood-vessels are beset with oil-globules. The intermediate connective tissue is proliferated, and an amorphous and nearly transparent substance is formed, in which crowds of nuclei are seen. We have therefore the symptoms of chronic inflammation of the neuroglia, and this may eventually creep up to the opposite column of the spinal cord, causing diffuse interstitial myelitis.

It is not yet settled whether these secondary lesions are owing to irritation spreading from the seat of the primary lesion, or to the withdrawal of the nervous influence of the ganglion cells of the grey matter of the brain upon the nutrition of the nervous matter below the lesion. Professor Westphal, of Berlin, has artificially produced a secondary degeneration of this kind in animals by destroying the corpus striatum and thalamus opticus, and dividing the cord; while Vulpian has seen negative results from the same operative procedures. In some cases senile atrophy of the entire cerebral matter, involving more especially the hemispheres, is seen to follow an attack of hæmorrhage.

Interstitial inflammation may spread eventually from the cord to the peripheral nerves. They become swollen, their tissue denser, and the several fibres are seen to be separated and compressed by an excessive formation of connective tissue. The bulk of the nerves may occasionally be felt increased during life. The muscles under these circumstances beccme pale, and waste away; the primitive fibres lose their stripes; oil-globules appear and the nuclei of the sarcolemma are multiplied. The joints are often painful a few weeks after the stroke; the synovial membrane is congested, its fringes hypertrophied; the quantity of synovial fluid is increased, and the tendinous sheaths become vascularised.

Dr. Scott Alison was the first to draw attention to the fact that secondary affections of the joints are not infrequent after

cerebral hæmorrhage. The joints mostly implicated are the knee, elbow, wrist, and ankle. They are hot and swollen, and pain is experienced not only on moving them but also when they are kept quiet. This affection is not connected with gout, rheumatism, or any other diathesis, but the direct consequence of the cerebral lesion; and always spares the joints of the healthy side. Charcot has seen a case in which there were deposits of urate of soda, either crystallised or amorphous, in the cartilages of the joints of the paralysed side, while those of the healthy side did not show any such alteration. According to the same observer, there is in these cases a regular synovitis with vegetations, proliferation of the nuclei and fibres which constitute the serous membrane of the joints, and increase of the size and number of the capillary vessels. These affections are distinguished from rheumatic affections by being limited to the joints of the paralysed limbs; the time at which they appear, in relation to hemiplegia; and by the co-existence of other trophic disturbances of the same character, such as decubitus. If the spine is affected, acute muscular atrophy of the paralysed limbs, cystitis, nephritis, etc., may be present.

Where the paralysis and contraction have lasted a considerable time, the joints undergo further degeneration, probably in consequence of their long immobility. The synovial membrane appears thickened, and there may be ulceration and formation of false membranes, and complete ankylosis. The limbs waste away in their entirety, and more particularly so in children, where the growth is arrested and the spinal nerves may become altogether wasted. The nails become yellow, crooked, and fissured; the hair thicker and longer; and the skin hypertrophied.

The late rigidity of the muscles, which is observed as a consequence of the pathological changes which have just been described, is different from the early rigidity, by appearing some months after the stroke, and by being progressive and permanent. It affects certain sets of muscles more than others. In the face the commissure of the lips is eventually drawn to the paralysed side, in the same way as is sometimes seen in old cases of paralysis of the portio dura, with consecutive contraction. Errors in diagnosis have been occasioned by this circumstance, for a hasty examination has sometimes induced

the practitioner to take the paralysed for the healthy side. The sterno-cleido-mastoid muscle may, by its rigidity, draw the head towards the shoulder of the paralysed side. But the muscles chiefly subject to it are those of the upper extremity, and amongst them again chiefly the flexors. The forearm is generally pronated, and where the contraction of the fingers is very great, the nails may penetrate the skin and cause ulceration.

On endeavouring to stretch the contracted fingers considerable pain is caused, and resistance encountered; sometimes the patient turns faint and sick during the time. As soon as the effort is discontinued, the fingers resume their previous position.

The faradic and galvanic excitability of the paralysed muscles is in the large majority of cases normal; and this serves as a good test, in addition to others, to distinguish cerebral from peripheral paralysis, lead-palsy, and other affections in which the electric contractility of the muscles is either greatly diminished or entirely lost. That this should be so is owing to the fact that trophic alterations of the paralysed muscles are on the whole rare in cerebral palsy. Even where this has existed for many years, there may be hardly any wasting of their substance, while in cases of injury to the motor nerves, in saturnine poisoning, and also in some forms of myelitis, viz. where the grey matter in the centre of the cord is suffering, rapid and extensive wasting of the muscles is encountered.

In some cases of cerebral palsy, however, we find great diminution of electric excitability, and we then have to trace this either to simultaneous affections of the intracranial trophic centres, or to Türck's secondary degeneration.

In other cases there is an increase in the galvanic and faradic excitability of the muscles, which is owing to chronic sub-inflammatory irritation of the nervous centres.

In paralysis of the portio dura from effusion into the pons Varolii there is generally early diminution of the electric contractility of the muscles.

Occasionally the paralysed nerves and muscles show the phenomena of convulsibility and exhaustibility. It is then noticed, that on first using the current, the contractility may

be either normal or diminished, but that, as the current continues to act, there is a rapid increase in the response of the muscles, which sometimes becomes most violent. In other cases the response of the muscles is at first normal, but diminishes considerably as the current continues to act. The two conditions may even occur together in the same case, so that on commencing to act we find convulsibility, which after a time is followed by exhaustibility.

The *mental functions* generally suffer in corpus striatum hæmorrhage; and as this organ is not the seat of these functions, we must assume that the deterioration of the psychical faculties is owing either to the shock received by the hemispheres during the attack, or to the secondary degeneration of nervous tissue which takes place after it.

Amongst the mental faculties, the memory is most apt to fail, and more particularly so for occurrences which have taken place since the attack. The temper of the invalid generally becomes peevish, irritable, and capricious. When atrophy of the hemispheres sets in, there is considerable mental decay, and at last complete imbecility.

The nerves of special sense are rarely much affected. There is sometimes deficiency of smell on the paralysed side, but this is more owing to paralytic narrowing of the nostril than to any affection of the olfactory nerve. A deficiency of taste which has sometimes been noticed is probably owing to affection of the chorda tympani, and not to anæsthesia of the glosso-pharyngeal or fifth nerve. The hearing is occasionally less keen. Optic neuritis is rare, but hemiopia somewhat more frequent.

Charcot and Veyssière have shown, both experimentally and clinically, that hemianæsthesia occurs after disorganisation of the posterior part of the internal capsule; but Dr. Ferrier[1] thinks that the cause of the hemianæsthesia in this case is not due to disorganisation of true centres of sensation, but merely to interruption of the path of transmission from the organs of sense to the sensory centres in the cortex, and these latter he believes to be the hippocampus major and the uncinate convolu-

[1] Loc. cit. p. 182.

tion. Destructive lesions of the hippocampal region have in his hands produced abolition of tactile sensation on the opposite side of the body.

A limb which has been deprived of tactile sensation becomes really inanimate, although there may be no motor paralysis. It may still be moved, but only under the guidance of the eye; and as soon as this latter is withdrawn, the condition of the limb for the time being drops out of consciousness. Thus, in Demeaux's case,[1] the woman moved her muscles by the commands of the will, but was unconscious of the movements which she made. She did not know in what position her arm was, whether flexed or extended. When she was requested to put her hand to her ear, she did so at once; but if the doctor put his own hands between the patient's hand and ear, she was not aware of it; nor did she know when her arm was prevented from carrying out a movement. If the doctor held the patient's arm on the bed, and then requested her to put her hand to the head, she made an effort towards it, but soon after ceased to do so, being under the impression that she had carried out the movement.

In these cases there is also a contraction of the field of vision, and difficulty to distinguish colours, on the side opposite to the cerebral lesion.

In most cases of corpus striatum hæmorrhage sensibility appears dull during the first few days after the stroke; but this is generally owing more to the torpor which exists at that time throughout the system than to any special lesion of sensibility.

Dr. Broadbent, who looks upon the thalamus opticus as the centre of conscious sensation, in the same way as the corpus striatum is believed to be the central motor ganglion for the opposite side of the body, explains the absence of permanent or even complete anæsthesia in most cases of cerebral hæmorrhage by the circumstance that sensation is altogether more easily transmitted, even by diseased structures, than motion. Dr. Brown-Séquard has experimentally shown that injury to the white motor columns of the spinal cord is at once followed by appreciable muscular paralysis, while considerable injury may

[1] 'Thèse des Hernies crurales.' Paris, 1843.

be done to the grey matter before any loss of sensation becomes apparent; and a certain degree of sensibility may persist in the whole of that part of the body behind the seat of the injury, provided only a slender bridge of grey matter is allowed to remain. Although it has not been experimentally demonstrated that this condition also prevails in the higher portion of the sensory tract, including the thalamus opticus, there would be no stretch to assume that this is so; from which it would be fair to conclude, that only such an amount of destructive change as would leave no fragment of the thalamus in connection with the lower sensory tract, would lead to complete anæsthesia.

The temperature of the paralysed limbs undergoes considerable changes during the progress of the disease. At first there is increased heat in the affected side, which generally amounts to 1° or 2°, but may be much higher in exceptional cases. This initial increase of temperature is no doubt owing to paralysis of the vasomotor nerves which course with the crura cerebri, and which must be affected by the lesion, provided this occupies the crura cerebri or the central ganglia. Where there is progress towards recovery, the temperature gradually becomes equalised in both sides; but where the paralysis persists, the affected limbs are eventually found to be colder than those of the opposite side. This diminution of temperature we have to explain by the diminished energy in the cellular changes going on in the tissues, and which are the ultimate source of the body-heat. Circulation is generally sluggish under these circumstances, and there is passive hyperæmia in the paralysed parts. The skin does not accommodate itself to varying circumstances; there is œdematous swelling in the arms and hand; the conjunctiva and the mucous membrane of the nose are congested, and secrete mucus more profusely than usual. There is sometimes a dull ache or violent pain in the paralysed limbs, which is increased by pressure; and when the patient attempts voluntary movements, there is often tremor which lasts as long as the effort.

Eulenburg has made sphygmographic researches on the radial pulse of the paralysed and healthy side, and found that there is diminution or loss of arterial tone in the paralysed limb. The artery no longer becomes expanded by the primary

wave, or by the succeeding secondary wave, nor constricted after these have passed off, so that both elevations and depressions are not well marked, and the whole tracing resembles the simple monocrotous pulse.

Dr. Broadbent has pointed out, that in that form of cerebral hæmorrhage which is owing to blood-poisoning from contracted kidney, and accompanied with hypertrophy of the left ventricle, the pulse is peculiarly large and hard; there is little pulsatile movement in the artery; the vessel slips and rolls about under the finger like a piece of thick whipcord, and it is difficult to control pulsation by pressure. We have, therefore, the long, hard, large pulse of arterial tension, which gives a sphygmographic trace of gradual ascent, low elevation, rounded summit, and gentle unbroken fall.

Coexisting with this is re-duplication of the first sound of the heart over the inter-ventricular septum, extinction of the first sound, and exaggeration of the second sound over the aorta. The re-duplication of the first sound is best heard with the double stethoscope, one mouth being placed over the right and the other over the left ventricle; and the phenomenon is due to want of coincidence in the contraction of the two ventricles. The contraction of the left ventricle is retarded by the extreme resistance to the entrance of more blood into the already distended arteries, and thus the right ventricle has a slight start in advance. The diminution or extinction of the first sound over the aorta is likewise explained by increased arterial tension, inasmuch as this latter prevents the left ventricle from throwing its contents rapidly into the aorta, the action being thus rendered more gradual.

In old cases of hemiplegia we often find convulsive seizures, and even true epileptiform attacks, or choreic movements and irregular contractions in the paralysed muscles, owing to sclerosis of the pons and medulla oblongata.

Relapses of the apoplectic stroke are frequent, inasmuch as the vascular lesions are progressive in their nature; but the popular notion that it is *the third stroke which kills*, is quite fallacious, for in a large number of cases the first proves fatal. It is, however, a fact that many patients succumb to a third attack. Others survive a third and even further strokes,

although in so enfeebled a state that they are left, as it were, only shadows of their former selves. There is gradual but plainly perceptible decay of the mental and physical faculties, owing to secondary atrophy. Senile marasmus thus becomes established, and as there is much less resistance to injurious external influences than there was previously, a slight incident, such as a cold affecting the bronchial tubes, or intestinal catarrh, will rapidly destroy life. The ultimately fatal result is often much accelerated by the enforced rest, the bad hygienic conditions in which these patients live, and by decubitus on the sacrum.

The effects of hæmorrhage into various portions of the cineritious substance of the brain, into the cerebellum, pons Varolii, the medullary matter of the anterior, middle and posterior lobes, and the medulla oblongata, will be considered when the diagnosis of the various forms of cerebral hæmorrhage comes under discussion.

2. *Hæmorrhage into the Membranes of the Brain, Meningeal Hæmorrhage.*

This form of hæmorrhage is not nearly as frequent as the one which I have just described. It may be caused by rupture of aneurisms of the cerebral arteries at the base of the brain; by cerebral hæmorrhage extending into the meninges; by a general hæmorrhagic diathesis, in which effusions of blood take place into diverse other organs besides the meninges; and by injury, such as a blow or fall, in consequence of which healthy bloodvessels are ruptured.

Meningeal hæmorrhage is frequent in newly-born infants, and seems to occur in them chiefly when labour has been tedious and instrumental delivery found necessary. By the application of the blades of the forceps the meningeal bloodvessels are squeezed and ruptured, just as the vessels of the skin and cellular tissue; and this is more especially the case where the head of the child is large. The forceps is, therefore, not an absolute boon in these cases, as children who have been delivered by its aid may subsequently become paralytic, epileptic, and imbecile. Sometimes, however, meningeal hæmorrhage is produced where labour has been easy, and it then seems to be owing to the

edges of the skull-bones being squeezed against each other, whereby the bloodvessels of the pia mater become ruptured.

The following was the proportion of deaths in children under five years of age in England and Wales, from 'apoplexy'—which in this instance we take, in accordance with clinical experience, to mean meningeal hæmorrhage—in the year 1872:—

	Males		Females	
	Under 5 years	All ages	Under 5 years	All ages
1872	394	5840	291	5953

While, therefore, generally speaking, the proportion of women dying from apoplexy is greater than that of men, it would seem that in the infantile period of life the relations are reversed, boys being more liable to die of meningeal hæmorrhage than girls. Dr. Braxton Hicks,[1] who has likewise noticed this peculiarity, explains the greater death-rate of boys than girls during and shortly after birth, by the fact that at full term the male is larger than the female, being on the average ten ounces heavier and half an inch longer. Moreover, the cranium is in boys more completely ossified, and the delivery of the head through a narrow pelvis more difficult than in girls, as it cannot be so easily moulded into shape. In consequence of this state of things the circulation in the uterus, placenta, and umbilical cord becomes more easily obstructed, causing effusion of blood in various organs, and also inspiratory efforts, by which fluids are drawn into the larynx and trachea, producing asphyxia.

The quantity of blood which is found effused in the meninges varies from a few drops to twenty ounces; but it is generally large. As a rule the blood is coagulated, and spread in all directions, either in the subarachnoid space or between the arachnoid and dura mater, and extends to the base and convexity of the brain, and to both hemispheres. Where the effusion has taken place between the arachnoid and dura mater, the blood accumulates on the tentorium and the base of the

[1] 'Croonian Lectures on the Difference between the Sexes, etc.' *British Medical Journal*, 1877.

skull, from where it may enter the arachnoid space of the spinal cord. In adults the convolutions appear flattened, and the cerebral matter anæmic; but such is not the case in infants, where abundant meningeal hæmorrhage may be found together with hyperæmia of the brain. Occasionally there is hæmorrhagic softening in the neighbourhood of the effusion.

The symptoms of meningeal hæmorrhage are more simple and less diverse in character than those of cerebral hæmorrhage, which is explained by the circumstance that the blood presses not upon certain circumscribed portions, but more or less upon the whole of the brain. As both hemispheres are compressed, there is no hemiplegia, or other special forms of paralysis, but muscular debility, which soon passes into complete relaxation and paralysis of all four extremities. Before paralysis sets in, there is sometimes rigidity or convulsions of the limbs. There is no localised anæsthesia but a generally diminished perception of impressions, which gradually deepens into stupor and coma; and this latter is in some cases the first symptom of meningeal hæmorrhage. Occasionally, however, there are such premonitory symptoms as headache and vertigo; and towards the end there is generally involuntary discharge of the urine and fæces. The respiratory movements then become stertorous, sighing and interrupted, showing that the blood compresses the medulla oblongata, which speedily results in asphyxia. Death generally takes place on the first day; but in exceptional cases patients survive two or three days or even more.

Infants in whom meningeal hæmorrhage has taken place *in utero* are either born dead or in a state of partial asphyxia, which soon ends in death. Sometimes the children remain alive for five or six days, but are in a state of excessive debility; they show the condition of apoplexy, cannot be roused, and those functions of life which are still exercised proceed with extreme sluggishness. At last vomiting and convulsions set in, and death follows more or less suddenly. In cases where the symptoms are protracted, we may assume that the hæmorrhage began previously to birth, and only tardily reached such a degree as to be incompatible with life.

Adults who die of meningeal hæmorrhage are generally habitual topers or drunkards, and suffer at the same time from

disease of the liver. That form of meningeal hæmorrhage which is known as *hæmatoma of the dura mater* will be considered in the fifth chapter under the heading of Cephalitis.

3. *Hyperæmia of the Brain, or Determination of Blood to the Head.*

This is another pathological state which may cause the condition of apoplexy. There are two kinds of hyperæmia, viz. the *active*, or arterial, and the *passive*, or venous form. We speak of active hyperæmia where a larger quantity of blood than usual is supplied to the brain and its membranes; while this passive congestion means that the supply of blood sent up to the head is not actually increased, but that the blood does not return freely from the brain through the jugular veins to the right side of the heart, and is therefore allowed to accumulate in the intracranial cavity.

Active hyperæmia occurs chiefly in simple hypertrophy of the left ventricle of the heart, without valvular disease. This condition occurs in gluttons, and likewise in persons who are habitually obliged to undergo great physical exertion, such as blacksmiths, navvies, &c. We also find it in contracted granular kidney, where the blood is poisoned through insufficient elimination of effete matters, and the coats of the arterioles and ultimately the left ventricle itself become hypertrophied. The left ventricle thus acquires an excessive impulsive power, which will lead to congestion of the brain, and indeed sometimes to rupture of its bloodvessels. Hypertrophy of the left ventricle in connection with disease of the aortic valves may also lead to it, although in many instances of this disease one condition compensates the other, the excessive force of the heart's action being counterbalanced by the imperfect closing of the valves; but where the two states do not strictly correspond, active hyperæmia of the brain will be the result.

Certain pathological conditions of the blood, such as anæmia, chlorosis, and hydræmia, are also instrumental in inducing active hyperæmia. The impoverished state of the blood causes a condition of irritable debility in the motor centre of the heart, and the distribution of blood is then carried out

in an irregular manner, the general result being determination of blood to the head, and chilliness of the extremities. It would, however, be erroneous to look upon all cerebral symptoms which occur in acute diseases, and amongst them more particularly upon somnolence and delirium, as arising from cerebral hyperæmia, and to treat the condition with bleeding and other active measures, as was the fashion formerly. We now know that cerebral symptoms in febrile diseases are generally owing to the high temperature of the blood circulating in the cerebral vessels, and to its altered composition. In acute mania, however, intense hyperæmia of the brain and its membranes is generally discovered.

Active determination of blood to the head is likewise caused by the habitual use of opium, morphia, and alcohol, which paralyse the vasomotor system of nerves. In some persons, a few glasses of wine are sufficient to cause such congestion, and for these, total abstinence from intoxicating beverages is imperative. Inflammatory conditions of organs in the neighbourhood of the brain, such as erysipelas of the face, mumps, quinsey, severe burns, and the sudden suppression of habitual hæmorrhages such as menstruation and piles, have a like effect. Violent emotions, more especially after meals, may cause such severe hyperæmia as to lead to sudden death. In such cases the irritation is propagated from the peripheral end of the pneumogastric nerve, or from the sentient nerves of the skin, or from the nerve-cells of the cerebral convolutions, to the vasomotor centre in the medulla oblongata. From there it spreads to the vasomotor nerves of the pia mater, and causes dilatation of the arterioles. Redness of the face and conjunctiva is generally observed at the same time, showing that the irritation has been transmitted to the fifth pair of cerebral nerves.

Passive or venous hyperæmia of the brain is induced where there is some impediment to the return of venous blood from the brain, in consequence of which the capillary vessels and the veins become gorged. This condition is observed in cases where the jugular veins are compressed by tumours in the neck, or the superior cava by tumour in the mediastinum; in newly-born infants, by constriction of the umbilical cord; in insufficiency of the tricuspid valve, and stenosis of the left

atrium, where at each systole the blood returns into the auricles and the cavæ. In such cases the jugular vein appears distended, and sometimes pulsating. Fatty degeneration of the heart, dilatation of the right ventricle without hypertrophy, certain diseases of the lungs, as emphysema, and pleuritic effusions, may also lead to it. It is true that the mechanical obstacle to the return of the blood which exists in all these conditions is sometimes counterbalanced by increased force of the right side of the heart; but in acute cases of such diseases there is no time for a greater development of muscular fibre in the right ventricle; while in chronic cases it sometimes fails to occur from the age or general debility of the patient. Sudden obliteration of an artery will cause partial hyperæmia in the neighbourhood; and cerebral tumours may lead to congestion, either by irritating the substance of the brain, or by compression of the veins. Chronic inflammation of the arteries and thrombosis of the veins have a like influence. Severe mental application, prolonged waking, more especially when nursing the sick; great muscular efforts, which augment the intra-thoracic pressure and prevent the emptying of the jugular vein—such as violent coughing, more particularly in whooping-cough, vomiting, straining, playing on wind instruments, and singing—are also liable to be followed by passive cerebral hyperæmia. The employment of compressed air in certain branches of industry has led to the same result in workmen habitually engaged in such occupations. In the last stage of fevers we find that failure of the heart's action, together with the alterations of the blood produced by the disease, and the approaching paralysis of the vaso-motor centre, tend to produce the condition which we are now considering. Where it is only produced just *ante mortem*, it is generally confined to the meninges and the superficial portions of the brain, while the true pathological hyperæmia affects chiefly the deeper portions of the encephalon.

A liability to determination of blood to the head seems to run in certain families, and is often associated with general plethora and free living. It occurs more in adults and the aged than in the young, and more in men than in women. The most powerful predisposing causes are, however, pericarditis and endocarditis.

Hyperæmia of the brain is generally less intense than that of other organs, as for instance the lungs and spleen; and important symptoms of disease may be caused by an apparently insignificant increase in the quantity of blood circulating through that organ.

The brain and its membranes are mostly affected simultaneously, in consequence of the same causes acting upon both parts, and it would be impossible to separate these conditions clinically. There are, however, certain sources of error which should be carefully avoided in forming an opinion. In the first instance it must not be lost sight of that different parts of the brain have a different vascularity. The grey matter is more vascular than the white, the corpus striatum more so than the grey matter, etc., and in childhood the entire organ is more vascular than in old age. Again, there is a simple post-mortem hyperæmia, produced after the body has been for some time lying on the back, from gravitation of the blood to the lowest level. This is chiefly seen in persons who have been of full habit during life, or where death has occurred from chest disease, or where the blood has remained unusually fluid, as after delirium and maniacal excitement. In other cases, on the contrary, it is found that all the symptoms of determination of blood to the head have been present during life, and yet the condition is absent on the post-mortem table. We may explain this by assuming that, where there was active congestion, the loaded arteries have, just previous to death, emptied their contents into the veins; or that the congestion has been relieved by transudation of serum through the coats of the capillary vessels, producing œdema of the pia mater and the brain; or that the blood may not have remained sufficiently fluid after death to show the condition of hyperæmia.

In general, however, we find the following post-mortem appearances:—the skin and cellular tissue of the skull are swollen, and on opening the skull a great deal of blood is seen to escape. The vessels of the diploë are congested, the sinuses of the dura mater distended, the bloodvessels of the pia loaded with blood. On incising the dura mater the brain protrudes through the opening like a rupture, and appears swollen and more voluminous than usual. The convolutions are not well defined;

the pia is not adherent, and may easily be lifted off from the surface of the brain. The grey matter appears dark red, or of a violet colour: the white matter is not actually red, but shows more coloration than in health. Where the hyperæmia has been very severe, as in infants just after birth, the white matter may appear almost as dark as the grey substance. On making a section, a number of small points are seen—the mouths of opened bloodvessels—from which drops of blood are seen to ooze more or less profusely. On microscopic examination the cerebral capillaries appear to be dilated, having often double their ordinary diameter. The hyperæmia is mostly general, but where the causes are limited to the sphere of certain bloodvessels, it may be local.

Where this condition has existed a long time previous to death, or where there have been repeated attacks of it, permanent lesions are apt to occur in the cerebral bloodvessels, from having been subjected to prolonged pressure. The large and medium-sized veins of the pia mater appear wider and tortuous, especially on the convexity of the brain; the smaller veins, the capillaries and arterioles, when examined by the microscope, are seen to have attained twice or even three times their original width. On section the coats of the capillary vessels are plainly visible to the naked eye (*état criblé* of the French observers). Sometimes the capillaries are ruptured, with consequent extravasation of blood; but more frequently the liquid constituents of the blood are effused through the coats of the bloodvessels, producing œdema of the brain and pia mater, and thickening of the arachnoid membrane. The brain itself ultimately wastes away, and its ventricles become dilated and filled with serum.

The symptoms of determination of blood to the head vary according to the causes which give rise to it, the degree of congestion which is present, and the extent to which it affects the different portions of the brain; but they are commonly characterised by being *multiple, not very severe, and lasting only a short time.*

In mild cases the symptoms are those of increased excitability, such as we would expect from slight compression of an organ which is enclosed in rigid walls. There is headache, in-

creased by the horizontal position; or a feeling of pressure and heaviness in the head, probably owing to compression of the dura mater, which is richly endowed with sentient fibres. There is also great disinclination to mental exertion, intolerance of strong light, noise, or of being touched; singing in the ears, sparks or black specks floating before the eyes; crawling sensations about the face and limbs, as of a procession of a thousand ants; drowsiness in the day, but restlessness at night, or sleep broken by vivid dreams or visions; grinding of teeth, and involuntary muscular jerkings. The face and conjunctiva are generally red, the head hot. The pupil is constricted through increased excitability of the third cerebral nerve. The heart's action is strong; the pulse large, hard, and slow, from irritation of the pneumogastric. Owing to this there is also often a feeling of sickness, and vomiting; and the bowels are confined. These symptoms last for a few days, and then disappear; but they are apt to return at any time, and may ultimately become habitual, more especially in hysterical, nervous, and alcoholised persons, and also in those who are subject to heart disease, and are of full habit. The slightest cause will then suffice to produce such attacks; for instance, some mental or physical effort, being in a heated room, in a theatre or concert, or 'dining out.'

When the degree of hyperæmia is more severe, additional symptoms make their appearance. There is giddiness, inability to stand, throbbing in the head, and mental disturbance. The patient is peevish and inclined to cry; there is delirium, or at least a rapid succession of foolish ideas, and incoherent chattering; the patient does not know where he is, throws himself about in a restless manner, is constantly in and out of bed, and walks from one room to another. There is inclination to run away: the face expresses terror; the patient hears voices which abuse and threaten him; he sees loathsome animals run about; and after a period of more or less prolonged excitement, ultimately falls into a deep sleep, which is accompanied by total relaxation of the limbs, and involuntary evacuation of the fæces and urine. On awaking there is deficient memory of what has occurred, great depression and prostration, and an imbecile countenance. Blenorrhœa of the conjunctiva and of

the buccal mucous membrane is often associated with it. Where these symptoms come on in the daytime, there is generally some immediate exciting cause, such as an annoyance or a quarrel.

Sometimes the principal symptoms of cerebral hyperæmia are delusions and hallucinations. Such was no doubt the state of Luther's brain, when, overcome by mental strain and excitement in his translation of the Bible, he hurled his inkstand at the apparition of the devil. Where cerebral hyperæmia has reached this degree, there is generally elevation of temperature, great thirst, loss of appetite, constipation, and the urine is scanty and loaded with lithates. The condition may last only for a very short time, and completely yield to rest and proper treatment; but where it becomes habitual, insanity, either in the form of melancholia or maniacal excitement, must be the ultimate result.

In other cases convulsions of an epileptiform character, with temporary loss of consciousness, may be observed. This seems at first sight strange, since we associate the occurrence of convulsions with anæmia rather than with hyperæmia of the brain; but it is capable of explanation if we consider that passive hyperæmia, when it has reached a considerable degree, really deprives the brain of useful arterial blood; and on the other hand, active hyperæmia, when severe, may lead to œdema, which renders portions of the brain anæmic.

The severest degree of hyperæmia of the brain causes the symptoms of apoplexy. Some recent authors have denied that such can be the case, and it certainly is less common than was formerly assumed; yet I do not entertain any doubt about its actual occurrence in practice. The capillary vessels and veins become sometimes so gorged with carbonised blood that no oxygen can reach the cerebral matter, and functional paralysis of the brain is the consequence. We find in such cases premonitory symptoms, viz., dizziness, pain and heaviness in the head, great indifference to the occurrences of daily life, and hyperæsthesia on the part of the special senses. All of a sudden the patient exclaims that the blood is rushing to his head, and he falls down in a state of insensibility. There is now more or less complete anæsthesia and paralysis. He may still

be roused by shouting to him, letting him smell ammonia, or dashing cold water in his face; but the perception of things in general is lost; there is difficulty of swallowing, the breathing becomes slow, stertorous, and intermittent, and the pulse quick, and so small as to be almost imperceptible. Reflex excitability persists, and may even be increased. In the majority of cases the patient rallies, and that much more quickly than would be possible after cerebral hæmorrhage; and there remains no decided loss either of motion or sensation. Sometimes, indeed, after consciousness has returned, the motor power may be enfeebled; there may even be hemiplegia, but this lasts only a few days, and disappears without leaving any traces behind. In a few exceptional cases, however, the symptoms gradually become worse; there is involuntary discharge of the excretions, and the patient dies in the deepest coma.

The symptoms of cerebral hyperæmia, such as I have just described them, are probably not so much owing to the increased intra-vascular pressure, as to the chemical changes which are going on in the blood during the time which the hyperæmia lasts. I have already adverted to the experiments of Pagenstecher (p. 99), who has shown that the intracranial pressure may be very much increased without the production of severe cerebral symptoms; besides which we have to consider that the blood, in the active as well as in the passive form of the disease, remains longer in the brain than is compatible with physiological laws. It thereby loses oxygen too largely, and becomes unduly charged with carbonic acid. Now we know carbonic acid to be a powerful excitant of the nerve-cells, and it appears natural that it should, when accumulated in undue quantity, cause symptoms of increased excitability of the brain. The symptoms of depression may be explained either by over-stimulation or by the transudation of serum which so often takes place under the influence of increased vascular pressure, producing cerebral œdema.

The progress of this disease is rapid where the hyperæmia is arterial; but in the passive form of it the symptoms are slowly developed. Sometimes nothing is complained of but sleeplessness, which may persist for years, before other signs of the affection make their appearance. Relief is afforded where a

collateral return of the blood from the head is effected; where the large veins dilate, and the right ventricle becomes hypertrophied; and finally by spontaneous hæmorrhage from piles and menstruation, and by augmented secretion of the bowels and kidneys. Actual recovery cannot be looked for unless the causes which have first led to the complaint should cease to act. Where death occurs from this disease we generally find that there is also pulmonary congestion. In seventy-nine cases of sudden death, the fatal issue was nine times owing to hyperæmia of the brain, and in six out of these nine cases congestion of the lungs was likewise present. Cerebral hyperæmia is not a very fatal disease, but repeated attacks of it lead to œdema, and subsequent wasting of the brain and pia mater, causing paralysis and imbecility.

4. *Embolism of Cerebral Arteries.*

This pathological event may give rise to the symptoms of apoplexy; but as it generally causes softening of the brain, with paralysis, I think it preferable to consider it in the next chapter under the heading of 'Paralysis.'

5. *Acute Alcoholic Intoxication.*

Everybody knows that an acute excess in alcoholic drink may produce the appearances of apoplexy; and the question, 'Drunk or dying?' is one that is only too often, in the streets of London, submitted to the discrimination of the police.

There are three degrees of acute alcoholic intoxication, in all of which the functions of the brain are considerably altered. The first degree, which is caused by imbibing a comparatively small quantity of diluted alcohol, is characterised by symptoms of cerebral excitement, which are mostly of a pleasant character, with subsequent slight depression, which is decidedly unpleasant. Persons in this condition are described not as 'drunk' but 'fresh;' as 'having taken luncheon' or been 'dining out.' The second degree, which is actual *drunkenness*, is produced by taking large quantities of diluted or undiluted alcohol; and the nervous system is then more deeply affected. After a period of excitement, which is of a variable character according

to the temper of the person concerned, and the kind of alcoholic stimulant which has been taken, the speech becomes indistinct; the gait tottering; memory and judgment fail; there may be maniacal excitement and delirium, followed by a profound and prolonged sleep, from which the toper awakes with symptoms of considerable brain-depression and acute gastric catarrh.

We are here, however, principally concerned with the third degree of alcoholic intoxication, in which a person is '*dead drunk*.' This occurs either subsequently to the second degree, or it may come on rapidly without premonitory symptoms of importance, after the ingestion of enormous quantities, mostly of raw spirits. This degree is characterised by the symptoms of apoplexy and collapse. The face is at first livid and swollen, and later on pallid and collapsed; the eyes are staring, fixed and glassy; the conjunctiva is injected; the pupil generally dilated, more rarely constricted, sometimes oscillating between dilatation and constriction. The heart's action is at first tumultuous, but later on weak and intermittent; the pulse is feeble, and often hardly perceptible. The skin is at first hot and streaming with perspiration, afterwards cold and clammy. Respiration is at first accelerated, but afterwards slow and stertorous, from paralysis of the soft palate. A viscid mucus is seen to flow from the mouth, and there may be vomiting of sour mucus and particles of undigested food smelling of alcohol. The muscles do no longer obey the orders of volition, and if the drunken man is still about, he staggers, and seems semi-paralysed. This muscular debility ultimately reaches such a degree that he falls down anywhere; and having entirely lost the sense of danger he may be frozen or burnt to death, or get drowned, or fall into a precipice. He is now in the condition of apoplexy. Speech, which was at first stuttering, is entirely gone; the organs of special sense have lost their power of responding to ordinary stimulation; reason, memory, and judgment are absent. Before the induction of coma, passion and brutality sometimes gain the upper hand, and acts of violence are committed, of which, if recovery takes place, the drunken man has no recollection, but which may nevertheless lead him to the scaffold. When coma has once set in, there is generally involuntary discharge of the urine and fæces, deathly pallor,

and general convulsions of the face and body. In cases which end fatally, the pupil becomes widely dilated, respiration rare and jerky, the muscles relaxed, the pulse imperceptible, and froth is seen issuing from the mouth. Cases of this kind in their severest form are seen in the London Docks, where men sometimes suck raw spirits through a straw from a cask until they become insensible. In such cases the specific gravity of the urine has been known to fall below 1000.

Death may occur within ten or fifteen minutes after the ingestion of enormous quantities of raw spirit ('twelve ounces of whiskey at a gulp'), and in such cases coma may set in directly without a previous period of excitement, or the unconsciousness may continue twelve or twenty-fours before death. Occasionally the drunken man has appeared to be in a fair state of recovery, when suddenly dyspnœa comes on, and ends in asphyxia; or he will sleep on heavily for many hours, and at last awakens deadly sick, with a splitting headache, a coated tongue, staring eyes, and all the signs of a severe acute gastro-intestinal catarrh, which may assume the form of cholera.

This is not the place for entering into the question of how we are to decide between the coma of drunkenness and the coma of cerebral hæmorrhage, which will be considered hereafter: suffice it to say that in some cases such a diagnosis is impossible during life, and can only be made on the dissection table. In death from acute alcoholism a strong alcoholic smell is observed in the cavities of the body and in the muscles. The liver, spleen, and kidneys are hyperæmic, the mucous membrane of the pharynx, œsophagus, stomach, small intestines, and bronchial tubes is red and injected. The membranes and substance of the spinal cord and brain are hyperæmic, the left ventricle and the arteries empty, the right side of the heart, the large veins and the tissue of the lungs contain a very large quantity of dark fluid blood. The brain is firm and white, and in the ventricles a quantity of serum is found which smells strongly of alcohol.

6. *Acute Intoxication by Opium and other Narcotic Poisons.*

This occurs by intentional poisoning and suicide, and likewise by taking or giving a medicinal overdose through mistake

or ignorance. The latter is often the cause of death in infants, whose mothers or nurses administer to them 'soothing syrups' and similar nostrums containing opium. Many deaths which are registered in the first few years of life must be attributed to this baneful practice. Children are known to be extremely sensitive to the influence of opium; and a case is on record where a dose of the compound tincture of camphor corresponding to $\frac{1}{90}$th grain of opium killed an infant one month old. For adults a fatal dose of opium is 20 to 30 grains, and of morphia from 3 to 6 grains. If subcutaneously administered, two-thirds of a grain of morphia have proved fatal.

The symptoms of opium poisoning are generally those of depression, not of excitement. In the lower animals, where the brain is only imperfectly developed, more particularly in frogs, convulsions are produced, and coma is not so pronounced, while in man stupor is the principal symptom. In infants, however, and also in the lower races of mankind, as in the Negros and Malays, convulsions are observed after its ingestion. Vertigo, fulness in the head, drowsiness fast developing into stupor, great itching of the skin, constipation, and an opiaceous smell of the breath and perspiration, are the principal symptoms in adults. Priapism, retention of the urine, and constipation distinguish this condition chiefly from cerebral hæmorrhage. In cases which are to end fatally, the muscles become completely relaxed, the pulse imperceptible, respiration arrested, and the body-heat lowered. Death may take place within an hour from taking the opium, but it generally occurs within six to eight hours. Occasionally, however, the case runs on for one or several days, and there may be secondary asphyxia, so that patients who appeared to be in a fair way towards recovery sink more or less suddenly with the symptoms of collapse. This is no doubt owing to further absorption of the poison, portions of which had not at first entered the circulation.

It would be extremely difficult, if not impossible, to find out how many deaths from 'apoplexy' which have been registered have really been owing to opium poisoning confounded with meningeal or cerebral hæmorrhage; but little doubt can exist that especially in former years this has not unfrequently been the case. Opium poisoning is now a special entry in the

Registrar-General's Reports, and showed the following particulars in the year 1874 :—

	Males	Females
Opium	13	11
Morphia	6	2
Laudanum and syrup of poppies .	39	22
Godfrey's cordial . . .	4	—
Chlorodyne	4	—

The same remarks apply to poisoning by other substances.

Hydrocyanic or *prussic acid* will cause sudden coma, with the symptoms of apoplexy, if very large doses, say one or two drops of the anhydrous acid, or one or two drachms of the diluted acid, are taken. Death is almost instantaneous under such circumstances. When the dose is somewhat smaller there may be coma lasting for a few minutes, and then ending in death. In still smaller doses there is a feeling of giddiness, tightness of the chest, and stertorous respiration. The breath smells of bitter almonds. Suddenly there is a scream, followed by convulsions, involuntary discharge of the urine and fæces, and loss of consciousness. The patient is now in a state of apoplexy. The pupil is dilated, the muscles relaxed, froth streams from the mouth, respiration and the heart's action become irregular, and at last extinguished. Death generally ensues within an hour from the ingestion of the poison.

Prussic acid is contained in the kernels of bitter almonds, peaches, apricots, plums, cherries, and quinces, and children have shown symptoms of poisoning after eating from four to six such kernels.

Poisoning by prussic acid is now a special entry in the Registrar-General's Reports, and caused, together with its congeners, the following mortality in 1874 :—

	Males	Females
Prussic acid	14	1
Oil of almonds	2	1
Cyanide of potassium . . .	7	3

Some other narcotic poisons produce the symptoms of apoplexy, but poisoning by them occurs so rarely that it need not occupy us here.

7. *Sunstroke.*

Sunstroke, or, more correctly speaking, heat-stroke, is essentially a disease of tropical climates, but it occurs also in the temperate zone, more especially amongst infants and young children, soldiers, and agricultural labourers. I have already adverted to the circumstance that for the production of heat-stroke in infants no direct exposure to the rays of the sun is necessary (p. 74); but in adults it scarcely ever occurs except during or shortly after exposure to a powerful sun, more especially when the system has been lowered by great fatigues and over-exertion. Marches of troops in the heat of the day; sleeping in the foul atmosphere of crowded and ill-ventilated barracks or cabins; the regulation dress of the soldier, including the stock, tight coat, and cross-belt, which impede the action of the lungs, and thus block up to some extent one of the depurating channels of the system—these and all other influences which debilitate the system, predispose to the occurrence of sunstroke.

There are two forms of this malady, one of which shows the symptoms of *syncope*, and the other those of *apoplexy*. The former of these is the most dangerous of the two; indeed, not unfrequently a man who is walking in the street or working in a field, and appears to be in perfect health, is seen to drop down dead. In some cases, however, certain symptoms precede the stroke, which show that the blood is becoming over-heated. The skin is hot and dry; the eyes look red and staring; and giddiness and pressure on the head are complained of; then there is a sudden faint, and death from cessation of the heart's action. In these cases the fatal result occurs so suddenly that treatment has no chance of saving life, more especially as the appliances, which would be of the first necessity, are generally not at hand.

The apoplectic form of the disease is not so rapidly fatal. There are the same symptoms which have just been mentioned

as premonitory of the first variety, in addition to which symptoms of mental derangement manifest themselves, which may last for several hours. There are delusions, hallucinations, and at last maniacal excitement, in which the patient may commit homicide or suicide. In such cases there is frequent micturition, and constipation of the bowels. After the stage of excitement has lasted for some time, a period of depression follows; there is great drowsiness, which at last merges into insensibility and apoplexy. Death takes place by the coma gradually deepening, or is preceded by an attack of general convulsions; but if the case be treated in time it may end in recovery, which is, however, often protracted for many weeks.

Patients who have once suffered from sunstroke seem never to regain their health entirely. They become subject to epilepsy, and complain of headache, impaired memory, and want of energy. Their temper is irritable, and they cannot bear any alcoholic stimulants, which must be looked upon as a positive poison for such persons. The skin, having once been paralysed, is often slow to recover its function, and the bowels remain obstinately confined.

The following appears to me to be the true pathology of heat-stroke. There is paralysis first of the heat-regulating centre in the cervical cord, and afterwards of the cardiac and vasomotor centres in the medulla oblongata. The first thing is that perspiration is arrested; and as the evaporation of sweat from the surface is the principal contrivance in the system for neutralising the effects of a high external temperature, and for equalising the body-heat, the immediate effect of its withdrawal must be unchecked influence of the external heat, to which is added the rise in the temperature of blood caused simply by loss of control over internal heat-production.

The unduly heated blood then becomes a poison for the cardiac and vaso-motor centres in the medulla oblongata, causing either of the two varieties of heat-stroke which I have mentioned, viz., syncope or apoplexy.

The remarks made about the registration of cases of opium poisoning as apoplexy, apply likewise to the relations of heat-stroke and apoplexy. Sun-stroke is, however, now a special

entry in the Registrar-General's Reports, and caused the following deaths in the three years from 1872–74:—

	Males	Females
1872	77	25
1873	71	25
1874	19	19

CHAPTER IV.

PARALYSIS.

THE mortality from paralysis closely approaches that from apoplexy. The following table shows the exact number of deaths from the various forms of paralysis which have been registered during six periods of five years each:—

Periods of five years	Deaths from Paralysis	Percentage of all Nervous Diseases	Percentage of General Mortality
1838–42	26,465	12·61	1·52
1843–46	—	—	—
1847–51	35,975	14·93	1·77
1852–56	42,044	16·28	2·02
1857–61	44,447	16·33	2·05
1862–66	51,301	17·50	2·14
1867–71	55,895	18·11	2·25
Total of thirty years	**256,127**	**16·31**	**1·95**

This table shows *the popular impression that paralysis has increased in fatality during the last decennia, to be correct, not only relatively to the mortality from nervous diseases, but also to the entire mortality from all causes.*

Up to the present time it has been believed that men are more liable to paralysis than women; but the following table shows that *the influence of sex on the occurrence and fatality of paralysis is only a slight one, and that, on the whole, females are rather more apt to die of it than males*:—

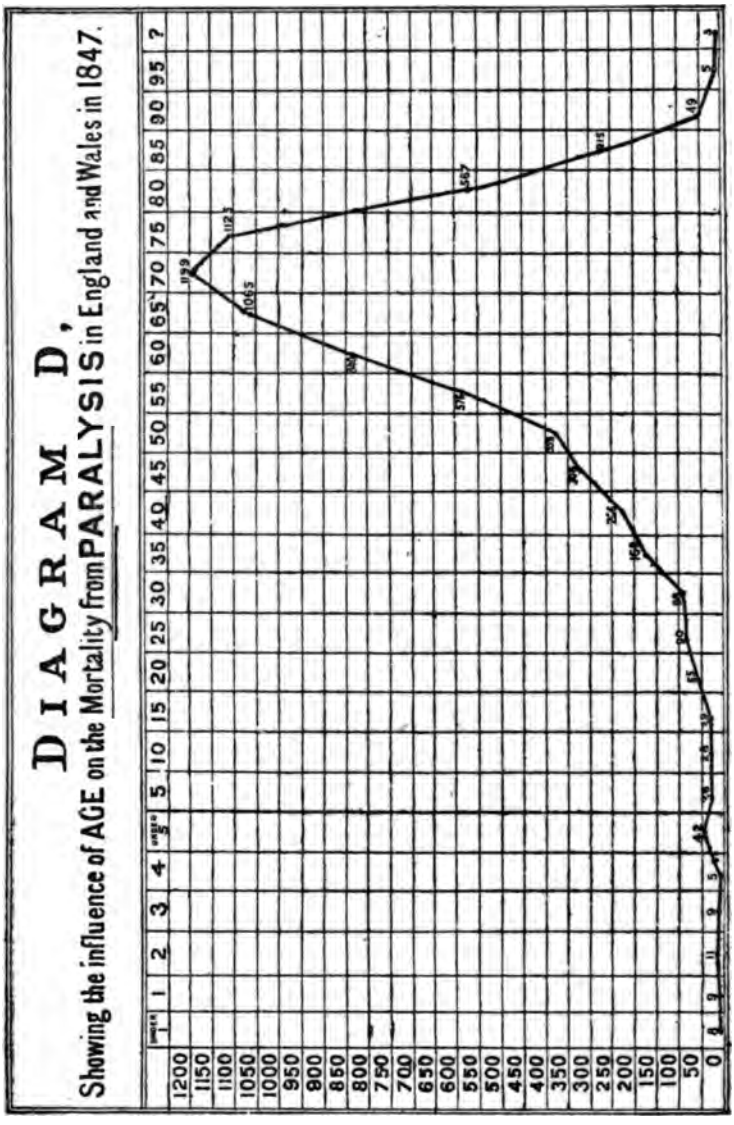
	Males	Per cent.	Females	Per cent.
1847–51	17,141	7·19	18,834	7·87
1852–56	20,169	7·81	21,867	8·47
1857–61	22,106	8·40	23,621	8·69
1862–66	24,987	8·77	25,754	8·84
1867–71	27,593	9·17	27,792	9·00

Having regard to the fact that there are more females in England than males, and that female mortality in general is less than male mortality, we find the following result for the year 1874:—

	Males	Females
Population	11,512,956	12,135,653
General mortality . . .	272,178	254,454
Death-rate in 1,000 . . .	23·6	21·0
Deaths from Paralysis . .	6,369	6,203
Percentage of general mortality .	2·34	2·42

While, therefore, the two sexes seem to die in nearly equal proportions of paralysis, we find that *age has a most powerful influence on the fatality of that disease.*

Paralysis, contrary to apoplexy, carries off but few victims in the earlier periods of existence; and this coincides with our clinical experience, which shows that that form of paralysis which is peculiar to infantile life, is but rarely fatal. The curve in the annexed diagram remains therefore low until the period of thirty-five years, when it points to 168 deaths. The rise is now pretty rapid, but more particularly so after fifty-five, when 576 deaths are recorded. The maximum of 1,199 is reached at seventy; at seventy-five the number is still very large, viz., 1,125. After that, however, there is a rapid fall, for at eighty, the deaths amount only to 567; at eighty-five there is a further great fall to 215, and at ninety to 49 deaths. Five are registered at ninety-five years, and three as occurring after that; yet considering how few people are alive after seventy-five, paralysis must be pronounced to be one of the most fatal diseases of old age.



The top line of numbers denotes the periods of age at which the deaths have taken place.
The side line of numbers indicates the numbers of deaths at the different periods of life.

The next diagram shows the deaths of males and females from paralysis at the different periods of life. The thin curve portrays these relations for women, and the thick one for men. It is seen that the two curves run a nearly identical course up to twenty years of age. At twenty-five the thick overlaps the thin curve, and continues to do so until forty-five; but at fifty the relations become reversed, and the thin curve keeps generally at the top from that time. Both reach the summit at seventy, but the thin apex is considerably higher than the thick, there being 616 female against 553 male cases. At seventy-five there is a slight, and at eighty a quick descent, but the difference between the sexes remains pretty much the same; the excess for women being 63 at seventy, 71 at seventy-five, and 63 at eighty. At eighty-five the difference is reduced to 39, and after that period both numbers are nearly alike.

Paralysis always proceeds from disease of the three great divisions of the nervous system, viz. the brain, the spinal cord, and the peripheral nerves; and we distinguish accordingly between cerebral, spinal, and peripheral paralysis. Of these three forms, cerebral paralysis is by far the most frequent, important, and fatal; spinal paralysis occurs much more rarely, and peripheral paralysis does not interfere with life, except in a few rare cases.

Cerebral paralysis may be caused by injury to the skull and membranes of the brain, more especially fracture of the skullbones, with penetration of foreign bodies. Exostoses, caries, and necrosis, and tumours pressing upon the brain from without, as well as the various inflammatory conditions which affect the membranes, may have the same influence. Hæmorrhage into the central ganglia of the brain, more particularly the corpus striatum, we have already seen to be a most frequent cause of paralysis. Hyperæmia of the brain, on the contrary, rarely causes paralysis, except where it leads to œdema with subsequent anæmia of the brain, as is seen particularly where it is complicated with chronic encephalitis and cerebral tumour. Encephalitis, which will be fully considered in the next chapter, may not cause paralysis if the degree of inflammation is slight, and the disease does not affect the neighbourhood of the central ganglia or motor convolutions; while palsies varying in degree will be brought about where the neighbourhood of the motor

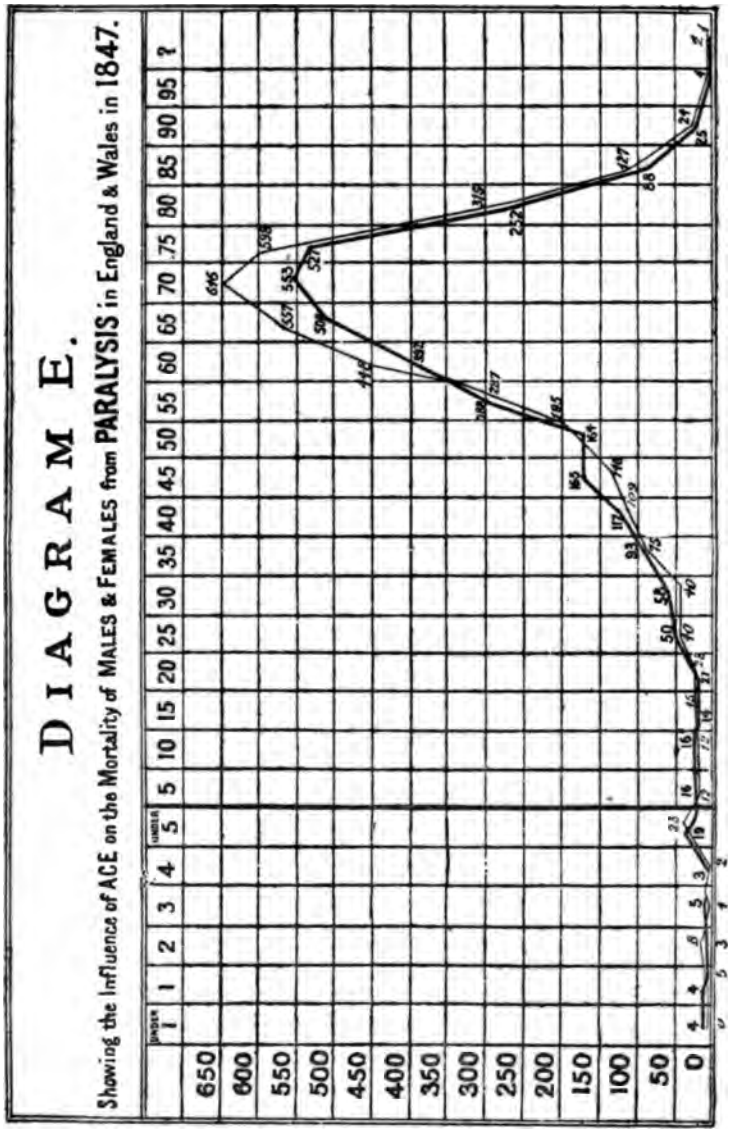


DIAGRAM E.
Showing the Influence of AGE on the Mortality of MALES & FEMALES from PARALYSIS in England & Wales in 1847.
UNDER 1
1
2
3
4
UNDER 5
5
10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
95
?
650
600
550
500
450
400
350
300
250
200
150
100
50
0

centres is invaded. Where encephalitis leads to cerebral abscess, the intracranial space is reduced, the capillary vessels of the brain are compressed, and portions of the brain rendered anæmic.

Sclerosis or proliferation and hardening of the neuroglia, with simultaneous wasting of the nerve-cells and fibres, causes peculiar forms of paralysis, which does not assume the form of hemiplegia, or even paralysis of one limb, but is unequally distributed over certain individual muscles. It is often seen that in the sphere of the same nerve some muscles have lost their power, while others continue in functional activity. This disease has a most insidious course, is often believed to be merely functional, and shows a tendency to progress downwards to the spinal cord, causing ill-defined forms of paraplegia.

Cerebral tumours may cause palsy by destruction or compression of the motor centres. They often lead to œdema, secondary inflammation, softening, and capillary hæmorrhage. Where, in consequence of the size of the tumour, the intracranial space is reduced, paralysis results from slowly-produced anæmia. It generally assumes the form of hemiplegia, which is apt to be associated with affections of the cerebral nerves. These are irritated or compressed by the growth of the tumour, whether the latter proceeds from the skull and the membranes of the brain inwards, or from the cerebral substance itself outwards. Occasionally hæmorrhage takes place in the tumour, and there may then be clinical symptoms of apoplexy from cerebral hæmorrhage, as described in the preceding chapter.

Softening of the Brain.

The most important cause of paralysis, apart from cerebral hæmorrhage, is, however, *embolism and thrombosis of the cerebral arteries, which lead to softening of the brain.* Softening was formerly believed to be an independent disease, which some pathologists thought owing to inflammation, while others considered it to arise from non-inflammatory disturbance of nutrition. The observations and experiments of Virchow and Cohnheim have, however, entirely transformed this department

of pathology, and given us a very complete insight into the morbid condition which is now under consideration.

The cerebral bloodvessels may be blocked either by small clots carried into them from a distance (embolism), or the clot may originate in the bloodvessel itself (thrombosis).

Embolism results most frequently from previous endocarditis, whether this be chronic or acute. In the acute form of the disease there is ulceration, and consequently embolism of capillary vessels may be caused; while in the chronic form warty excrescences are formed on the valves of the heart, more especially of the left ventricle, leading to insufficient action of the valves and stenosis of the ostia. Portions of these excrescences may be detached from their seats in the pulmonary veins, the left side of the heart, and the aorta, and carried with the current of the blood into the carotid or vertebral arteries, and from there into a cerebral artery. They generally follow the *left* rather than the *right* carotid artery, on account of the anatomical peculiarities of these bloodvessels, and go even more rarely through the vertebral than the right carotid. They proceed as a rule unchecked in their course beyond the circle of Willis, but become impacted in the mid-cerebral artery. Other vessels which are also liable to be obstructed are the internal carotid, the profunda, the basilar and vertebral artery, and the artery of the corpus callosum. Emboli consist of clots of blood, fibrine, proliferated connective tissue, and chalky concretions.

Embolism may also come on without previous endocarditis after myocarditis; from fibrinous coagula in aneurismal sacs; in abscess and gangrene of the lungs, and altogether in conditions where the activity of the heart is considerably lowered, which leads to the formation of clots in its cavities. This occurs chiefly in the later stages of tuberculosis and cancer, in empyema, and fatty degeneration of the heart.

Thrombosis is either venous or arterial. Coagulation of the venous blood is apt to occur when the power of the heart is diminished, and the circulation has consequently become sluggish. As this is more particularly seen in the condition known as marasmus, viz. in the advanced stages of phthisis and cancer, after febrile diseases, and chronic inflammation of the

joints, Virchow has termed it *marantic thrombosis*. Cardiac debility is in such cases combined with general anæmia and atony of the coats of the bloodvessels; and the thrombus is formed behind the valves of the veins, in the angle in which the valves adhere to the coat of the bloodvessels. When the vessel has lost its tone, the valves do not adhere properly, whereby the movement of the blood is impeded, and part of it retained. A slight coagulation then takes place, which rapidly increases by further deposits of fibrine from the blood; it soon fills the valvular sinus, projects beyond the edge of the valve, and proceeds in a direction towards the heart. After a time the vein becomes more and more constricted, and at last completely blocked up.

When a thrombus has been formed, parts of it may be detached, and carried forward with the stream of blood, until they become fixed in remote parts of the vascular system. Those thrombuses which are formed in the venous blood, and the right side of the heart, are carried into the pulmonary arteries, while those formed in arterial blood, viz. the pulmonary veins, the left side of the heart and the large arteries, may be carried into any portion of the arterial system of the body.

Plugging of the cerebral arteries by degeneration of their coats, and consequent thrombosis, is rare; it is chiefly found in the aged, and connected with general decrepitude. The principal changes in the arteries which give rise to it, are fatty degeneration, sclerosis, ossification, and deposits of chalk in the coats of the bloodvessels. All these changes tend to diminish the elasticity of the arteries, and their diameter is at last considerably narrowed. The condition is frequently associated with atheroma of the coronary arteries of the heart, which leads to impaired nutrition of that organ, diminished energy of its action, and sluggish circulation of the blood in the cerebral vessels, whereby the process of thrombosis in them is promoted. Thrombosis of the cerebral arteries is observed, apart from old age, in syphilis, pressure from tumours and effusions, leukæmia, septicæmia, in the puerperal state, in diffuse suppuration, caries, and after acute diseases and exhaustive discharges.

Ligature of the carotid artery for aneurism rarely causes softening of the brain. The thrombosis which is produced in

the bloodvessels by this operative procedure, extends generally only to the division of the common carotid, or at most to the circle of Willis, so that there is no impediment to the speedy establishment of collateral circulation. If thrombosis, however, proceeds beyond the circle of Willis, necro-biotic softening is the inevitable consequence. Occasionally a thrombus breaks up into several pieces by the current of the blood, each of which may penetrate into a cerebral artery, and thus cause several centres of softening.

Thrombosis of the veins and of the sinuses of the dura mater may occur in consequence of injuries to the head, and otitis with caries of the petrous portion of the temporal bone, or it may come on spontaneously. In the former class of cases there is generally antecedent phlebitis, while in the latter the structure of the veins is not primarily altered, and the cause is marantic, i.e. there is great cardiac debility with its well-known consequences. As the sinuses of the dura mater have rigid walls, and are traversed by layers of connective tissue, the occurrence of thrombosis in them is considerably facilitated.

When an artery has been plugged, either by a travelling embolus, or a thrombus, serious disturbances in the nutrition of the parts supplied by that bloodvessel are the inevitable consequence, unless collateral circulation can be established in the vascular sphere of the plugged vessel. The brain matter being particularly vulnerable, necro-biotic destruction of the same is the necessary consequence when it has for some time been deprived of nutritive material.

Heubner and Cohnheim distinguish a *basal* sphere of nutrition, providing the central ganglia and the mesocephale, and a *cortical* sphere of nutrition, the arteries of which supply the cineritious substance and the adjacent medullary matter. The mid-cerebral artery, which is the principal victim of embolism, provides the entire lenticular nucleus, part of the corpus striatum, the external capsule, and the anterior peduncle of the internal capsule, while its terminal branches reach the second and third frontal convolutions, those parts of the central, parietal, and temporal convolutions which are turned towards the convexity of the brain, and the insula. Necro-biosis in the sphere of this artery therefore leads most commonly to hemiplegia.

If the obstruction in the cerebral bloodvessels occurs on the near side of the circle of Willis, the nutrition of the brain does not generally suffer, because collateral circulation is quickly established; but if the obstruction is on the far side of the circle, necro-biosis is speedily established. The first event is anæmia of the parts supplied by the artery, without softening. This stage probably lasts only one or two days, as the brain matter is easily starved, and further changes are now produced; viz., either hyperæmia with œdematous swelling and hæmorrhage; or neither hyperæmia nor hæmorrhage, but yellow, or white softening.

Where the first of these eventualities takes place, viz., hyperæmia, œdematous swelling, and hæmorrhage, we have the condition known as *red softening*; and this chain of events has given rise to the theory of inflammatory softening, which was so long held by the French school. The bulk of the cerebral matter appears increased, while its consistency is diminished. The hæmorrhage from the capillary vessels is sometimes so extensive that the parts look as they do in cerebral hæmorrhage from the rupture of miliary aneurisms. The colour may vary from a bright pink to dark red, but as time goes on, generally within three or four weeks, it fades away, and the parts now present the appearance of *yellow softening*. This change in the colour is owing partly to the changes occurring in the effused hæmatine, and partly to the development of fat, which appears in connection with the retrogressive metamorphosis of tissue.

In red softening the microscope shows at first nothing but red corpuscles, and later on gradual degeneration of the nervous elements; granular globules are formed from the nuclei of the neuroglia, the cells of the capillary vessels, the adventitious tissue, the nuclei of the muscular coat of the bloodvessels, the connective-tissue-nuclei of the perivascular lymphatic spaces, and the spindles and ganglionic cells of the cerebral substance; and ultimately nothing is found but fatty detritus mixed with crystals of hæmatine.

Cohnheim has given the following rationale of this process:—Physiological experiments show that, when a terminal artery which has no further communication with any other arteries, is

blocked up, there is a reflux of blood from the proximate artery which is still pervious, into the corresponding vein and the entire stagnating vascular sphere belonging to the plugged artery. The consequence of this is hyperæmia and hæmorrhage by diapedesis of the red blood-corpuscles. As the blood cannot circulate in the artery, the coats of the bloodvessels are altered, and the blood-corpuscles transudate.

If the patient survives a sufficient time, *white* softening ultimately becomes produced. The brain matter is now changed into an emulsion of milky appearance, i.e., a liquid with solid particles floating in it. Sometimes a cyst is formed which is filled with liquid, and resembles the cysts which are found after an attack of ordinary cerebral hæmorrhage.

The second eventuality is *primary yellow softening*, without hyperæmia and hæmorrhage. This is simple necro-biosis, the parts undergoing at once fatty degeneration, without any other previous alterations.

Cohnheim has also shown experimentally under what circumstances no hæmorrhage, but primary yellow or white softening, is produced. It occurs where the blood coagulates so quickly in the entire vascular sphere of the plugged artery that any reflux of blood through the vein becomes impossible. In practice it is found that simple necro-biosis, or yellow softening, without hyperæmia and hæmorrhage, occurs chiefly where the functional energy of the heart is considerably below par.

The symptoms of softening of the brain vary considerably according as they are produced by an embolus or a thrombus. Their invasion is always much more sudden in embolism than in thrombosis. The symptoms of embolism are owing partly to the shock caused to the brain by the sudden entry of a foreign body, and partly to the anæmia which we have seen to be the immediate consequence of such an event. When the detached embolus settles in the mid-cerebral artery, there is for a short time vertigo and headache, and then all the symptoms of apoplexy, as seen in corpus striatum hæmorrhage. The apoplexy from embolism however lasts, as a general rule, not so long as that from rupture of miliary aneurisms in the same portions of the brain; and in many cases there is only sudden

hemiplegia without loss of consciousness. Where however there is apoplexy, it differs in no way clinically from the description which has been given in the previous chapter. It was formerly believed that we could distinguish the two kinds of apoplexy by the different colour of the face, a different behaviour of the pupils, the absence or presence of vomiting, and some other signs; but more ample experience has shown that all symptoms which have been described may be present in either of the two forms of apoplexy. Sometimes convulsions are observed, generally in the form of a regular epileptic fit, affecting all the muscles of the body, while in other cases the convulsions are confined to that side which is afterwards found paralysed. Occasionally the paralysis is incomplete, there being only weakness in the muscles of the face, arm, or leg, or impaired speech. When the embolus has blocked the basilar artery, vomiting is present. The left mid-cerebral artery being most commonly the seat of embolism, aphasia is connected with hemiplegia, as anæmia and softening of the third left frontal convolution (p. 51) is the result. When the central artery of the retina, or the ophthalmic artery are plugged by an embolus, sudden amaurosis is observed. Embolism of the mid-cerebral artery causes the ophthalmic artery to be gorged with blood; the papilla then appears red, and there is arterial and venous hyperæmia of the retinal vessels.

The symptoms of paralysis from embolism of the cerebral arteries may vanish more or less suddenly; and in such cases we may be certain that the attack has not been one of cerebral hæmorrhage, and that the changes in the brain induced by the embolus have only amounted to anæmia, and not to softening. Such recovery can therefore, in the nature of things, only occur a day or two after the stroke; for if necro-biosis is allowed to commence and to proceed unchecked, the symptoms of paralysis must be more permanent. After ligature of the carotid artery there has occasionally been hemiplegia of a few hours' duration, and which disappeared as soon as collateral circulation had been established. If the latter is imperfect there is proportionate improvement in the paralytic symptoms. The arm and leg may regain a degree of voluntary motion, but nevertheless remain useless for practical purposes; the speech returns but is

imperfect, and the mental functions, though not destroyed, are impaired. Where the lesion is extensive, death may be the consequence, but this is never sudden. It may occur twelve hours or later after the commencement of the symptoms, and is not unfrequently preceded by pneumonia, and a great elevation of temperature.

In general there is no initial decrease of temperature in softening, such we have seen to occur in cerebral hæmorrhage (p. 102); or if it should occur, it is much less marked. Bourneville has shown that in many cases of softening soon after the attack, the temperature rises suddenly to 102° or even 104°; it then falls again, reaches the normal average, and shows irregular oscillations. It sometimes remains stationary for a couple of days, or shows morning or evening falls. In cerebral hæmorrhage, on the other hand, the temperature, when it has once reached 102°, does not go back to the physiological standard unless a fresh effusion of blood should take place; and the oscillations are more regular, and occur in a narrower compass, than in softening. After the temperature has been stationary in softening for a more or less considerable time, it begins gradually to rise, and reaches 103° and 104°; but towards the end it is not nearly so high as in cerebral hæmorrhage, where it sometimes reaches 108°. After death there may be a slight increase, but as a rule the temperature falls more rapidly than it does in cerebral hæmorrhage.

In hemiplegia from embolism of the mid-cerebral artery, the paralysed muscles are also liable to contraction, although not so frequently as after cerebral hæmorrhage; and the character of these contractions appears to differ somewhat according to the cause of the lesion. In a case at present under my care at the Hospital for Epilepsy and Paralysis, Regent's Park, in a girl aged 17, the contraction of the paralysed flexor muscles of the arm, after embolism from endocarditis, presents this peculiarity that it is chiefly marked when the hand is touched; while when the hand remains undisturbed, it is well open. I have noticed this peculiarity in several similar cases, but whether it will be eventually found a distinguishing symptom between hemiplegia from embolism, and from cerebral hæmorrhage, will have to be settled by future experience.

Convulsions in the paralysed limbs, which ultimately develop into epileptiform seizures, are occasionally observed after the paralysis has lasted for some time.

Where smaller arteries are obstructed by embolism, there is only partial or incomplete paralysis, or no palsy at all. This is explained by the circumstance that only very limited areas of the brain are deprived of blood, and that collateral circulation is more easily established. If portions of the brain which do not belong to the motor centres are rendered anæmic and softened, of course no paralysis is produced.

The further course of the disease is determined by the extent of the necro-biotic process, which is often progressive in its character; and a number of fresh cerebral symptoms may therefore make their appearance as time goes on. Treatment may incline the balance to improvement or further decay; for as the establishment of collateral circulation affords the only prospect of a cure, the treatment must be tonic and stimulant, while lowering measures, as suggested by the old notions of inflammatory softening, are invariably followed by the worst results.

Virchow's and Cohnheim's researches on embolism would have been impossible without the aid of vivisection; and as a great improvement in our mode of treating such an important disease as softening of the brain is directly owing to these researches, the importance of vivisection for practical medicine, which has been denied by a set of ignorant fanatics, is by this fact alone clearly established.

The symptoms of cerebral thrombosis are more gradually developed than those of embolism. There is as a rule a large number of premonitory symptoms, which are owing to the gradual constriction and plugging of the artery. These are headache, vertigo, stammering, impaired memory, numbness and chilliness in one side of the body, local palsies, especially of ocular muscles, tottering gait, contractions of fingers, incontinence of urine, and other symptoms which have been frequently described as the initial mementos of 'softening of the brain.' Finally there may be hemiplegia, which is either gradually developed or occurs suddenly, when the vessel is completely blocked up; and this is either followed by death or

improvement. In the latter case the patient may remain tolerably well for some time; but the attack is likely to be repeated, as the causes continue to act. The symptoms vary according to the portion of the brain which is the seat of these retrogressive changes. Where the left mid-cerebral artery is plugged, aphasia and right hemiplegia will be produced, while if the same artery suffers on the right side, there is left hemiplegia without aphasia. Where a considerable number of small arteries is plugged, or where there is gradual and progressive thrombosis of large vessels, as is so frequently seen in the aged and decrepit, there is gradually progressing paralysis accompanied with muscular contractions, and impairment of the mental faculties.

Marantic thrombosis of the sinuses of the dura mater is often seen in strumous infants, who suffer from exhaustive diarrhœa. The symptoms in these cases are generally those of infantile cerebral anæmia or hydrocephaloid, as described in the second chapter. Rigidity of the muscles of the neck, back, and extremities is the rule, and these are soon followed by somnolence and coma. Nystagmus, strabismus, and ptosis are likewise occasionally present. The principal seat of the thrombosis in these cases is the sinus longitudinalis superior.

In adults the symptoms are sometimes ill-defined. There is great depression and apathy, headache, combined with sickness and vomiting; alterations in the size of the pupils; clonic convulsions and tremor of the limbs. In some cases the external veins which communicate with the sinuses are seen to swell. When the *sinus longitudinalis superior*, which communicates with the veins of the nose and the upper portion of the skull, is affected, there is epistaxis and dilatation of the veins on the temples and ears. In thrombosis of the *sinus transversus*, local œdema behind the ear may be encountered, from plugging of a vein proceeding from that sinus and the mastoid process. In a similar manner there may be hyperæmia of the fundus of the eye, exophthalmus, and œdema of the eyelids in thrombosis of the *sinus cavernosus*, which communicates with the ophthalmic veins.

The phlebitic form of thrombosis is found after otitis, with caries of the petrous portion of the temporal bone, and may

lead to meningitis and cerebral abscess. In phlebitis of the sinuses there are symptoms of septicæmia, such as rigors, a typhoid condition, dry tongue, loss of appetite, delirium, somnolence and coma. If paralysis or convulsions occur, they must be ascribed rather to the meningitis and abscess of the brain than to phlebitis of the sinuses.

I now proceed to consider the more important forms of paralysis separately.

1. *Aphasia.*

Loss of intelligent language was at first called 'aphemia' (Broca), but Trousseau, on the advice of Krisaphis, a learned Greek, introduced as more classical the term aphasia, from privative alpha, and φασις, speech. We have seen in the first chapter that the nervous contrivances for the mechanical execution and connection of the movements of articulate speech are situated in the medulla oblongata, pons, and corpora striata; while the intelligent formation of syllables, words, and sentences, or intelligent language (logos), resides in a definite portion of the cortical substance of the hemispheres (p. 51).

The principal current of speech goes through the left hemisphere, while a weaker, collateral current goes through the right hemisphere. This greater development of the left hemisphere for speech co-exists with the right-handedness of most people—that is to say, the left hemisphere is, during the period of infancy and childhood, chiefly trained for speech, as well as for combined voluntary movements. That the left hemisphere is not the exclusive seat of language is shown by the fact that, after destruction of the area for language within that hemisphere, emotional language and the faculty to use interjections is still possible. Thus most persons suffering from aphasia are still able to swear, or at least to say 'yes' or 'no,' while utterly unable to converse. There may therefore be loss of language without complete loss of words. This has led Dr. H. Jackson to think that the automatic faculty to speak words resides in the right, and actual intelligent language in the left hemisphere; but this seems a somewhat forced explanation of the facts which come clinically under observation. There can be no doubt that emotional excitement is more powerful and more

widely spread than that which is induced by thinking and imitation. It therefore not only implicates the motor centres in the brain, but also the spinal and sympathetic ganglia. In children, roughs, and savages the emotional language is always accompanied by pantomime; and as gestures are made with either or both hands, we must conclude that emotional excitement may lead to movements presided over by both hemispheres. Simple analogy would therefore show that the language of the affections and the faculty of using interjections are as much bound to both hemispheres as pantomime.

It is well known that facial expressions, such as laughing or crying, may continue to come on emotionally when they can no longer be produced at will; and under such circumstances the slightest cause is apt to give rise to them. The person so affected has a silly appearance, owing to the removal of the influence of the higher centres. An analogous case is that of the decapitated frog, in whom spinal reflex actions are produced in the wildest manner. The language of music, which is entirely one of sentiment and emotion, may persist after the loss of intelligent language. Béhier has recorded the case of a man who could say no other syllable but 'tan,' but who could still sing the two French national songs—viz. the 'Marseillaise' and the 'Parisienne,' but without singing any other words in the text except 'tan.'

The history of the localisation of intelligent language is identified with the names of Gall and Spurzheim, Bouillaud, Dax, and Broca. Gall,[1] in whose great work much that is sound and new is given side by side with fantastic theories which have obscured his otherwise well-deserved fame, found the seat of language in that part of the anterior lobes of the hemispheres which is situated above the orbital plates. Bouillaud[2] likewise located the faculty of the formation and memory of words in the anterior lobes, as he had found that loss of language and memory of words was the inevitable consequence of disease of the anterior portion of the brain. Dax[3] showed in

[1] 'Anatomie et Physiologie du Système Nerveux.' 4 vols., Paris, 1810–19.

[2] 'Traité de l'Encéphalite.' Paris, 1825.

[3] 'Lésions de la Moitié Gauche de l'Encéphale.' (Being a paper read in 1826, before a medical meeting at Montpellier.)

a remarkable paper—which was lost to the world until its republication by his son in 1861—that the left anterior lobe was the seat of language, and that therefore hemiplegia of the right side was generally combined with loss of language. It was, however, chiefly Broca[1] who gave precision to this doctrine, and laid down the principle that the left hemisphere is specially trained by education in early life for speech as well as for the more subtle kinds of work; that this is the cause of the dextral pre-eminence which obtains in the vast majority of men; that the posterior part of the third left frontal convolution has a special importance for intelligent language; and that most men, and all right-handed men, during their educational development, train only that convolution for the verbal expression of thoughts and ideas.

Broca's first proposition, that the third left frontal convolution has never yet been found diseased without there having been during life loss or impairment of intelligent language, has remained uncontradicted up to this time; but his second statement, that aphasia never occurs unless with disease of this convolution or those portions of white matter by which it is connected with the parts below, has not stood the test of time. Nevertheless it is unquestionable that in the vast majority of cases of aphasia disease of that convolution is discovered.

Thus in fifteen post-mortem examinations of cases of aphasia in Paris, Broca's convolution was found diseased fourteen times, while in one case (Charcot's) the left insula and the left parietal lobes were extensively softened, and the only sign of disease in Broca's convolution was fatty degeneration of the capillary vessels. Such cases, however, are exceptional. Many of those in which no disease was discovered in the left side of the brain have been described by men who were ignorant of the theory, and therefore paid no particular attention to the region involved. Dr. William Ogle[2] has recorded a typical case of aphasia, in which there was a lesion in the exact spot fixed upon by Broca, but which would certainly have escaped notice unless it had been specially looked for. The brain looked healthy on its surface, and it was only on removing the pia

[1] 'Bulletins de la Société Anatomique.' Paris, August, 1861.

[2] 'St. George's Hospital Reports.' London, 1867, vol. ii. p. 105.

mater that the almost diffluent condition of that limited spot was discovered. In this case there had been first rheumatic fever and valvular disease of the heart; later on, formation of fibrinous clots on these, and consequent embolism of a branch of the left middle cerebral artery, which contained a hard shotty bit of fibrine, completely obstructing the passage, so that when water was injected into the vessel it could not pass, although considerable force was used.

A second consideration is, that in exceptional cases not the left but the right cerebral hemisphere is trained during the educational development for language; and this appears to coincide with left-handed pre-eminence. Thus Moreau, of Tours, has related a case in which the whole third left frontal convolution was congenitally absent. The woman, however, could speak and read very well; she was able to sew with the left hand, and she had evidently during childhood trained the right cerebral hemisphere for language and the finer kinds of movements, on account of deficiency of the left.

A third consideration is, that not unfrequently during life mistakes in the diagnosis of the disease are made. Not everything that has been said to be aphasia has, on stricter scrutiny, turned out to be aphasia. Dr. John Ogle once had a case brought to him as one of aphasia, in which, however, it was found on examination that the patient had no tongue, and could therefore not articulate properly. Dr. Morell Mackenzie has informed me that cases of aphasia are occasionally sent to him for examination by the laryngeal mirror under the impression that the patient has simply lost his voice; and I well remember that in a discussion on this subject at one of our learned societies some years ago, an esteemed physician, since deceased, complained bitterly that the good old word 'aphonia' had recently been dropped for the new-fangled term 'aphasia,' the use of which he was utterly unable to understand!

The most convincing cases are those in which the lesion is limited; in which there is no other disturbance of health; and in which aphasia has been quickly and permanently developed. Of such cases there is at present no lack. A boy, aged five, who was a great chatterbox, fell out of the window and injured the left frontal bone, which was found depressed. There was

no hemiplegia or other kind of paralysis, but the boy had entirely lost his language. The wound healed in twenty-five days, but the child, although intelligent, remained dumb. A year afterwards he was drowned, and at the autopsy a cyst of the size of a filbert was found in the left third frontal convolution and the adjacent portion of the left frontal lobe.

A man fell with his horse, but got up, took hold of the reins, and wanted to jump into the saddle, when a physician who happened to accompany him expressed the wish to make an examination. It was then found that he could not speak, but had to make himself understood by pantomime. A small wound in the left side of the head was found, with depression, but there was no paralysis of any sort. The patient ultimately died of purulent meningitis, and the autopsy showed that a fragment of bone had penetrated into Broca's convolution, which, together with the second left frontal and the island of Reil, was found softened.

Besides cases of localised injury those instances are chiefly important in which we find necro-biotic softening from embolism or thrombosis of small branches of the middle cerebral artery. A large clot of blood, a cerebral abscess, tumour, and advanced sclerosis of the brain, do not allow of very convincing conclusions. Embolism and thrombosis of the middle cerebral artery or its branches are found to be the most frequent causes of aphasia. Jaccoud has attributed this to the more frequent occurrence of such a lesion altogether, but other cerebral arteries are nearly as liable to the disease as the one just mentioned.

In order to subvert Broca's theory it would be necessary to bring forward a thoroughly well-described case of sudden destruction of the third left frontal convolution in a right-handed, or of the right frontal convolution in a left-handed person, without aphasia; or destruction of both frontal convolutions should be shown at the autopsy of a person who had been able to talk intelligently until death. As yet this has not been done, and a large number of indisputable facts make it most unlikely that it ever will be done.

The frequent coincidence of aphasia with right hemiplegia has been disputed, but unsuccessfully. Dr. Séguin, of New

York, found that in 260 cases of hemiplegia and aphasia, the left hemisphere suffered in 243, and the right only in 17. It has been said that lesions of the left hemisphere are altogether more frequent than those of the right; but Charcot and Vulpian have shown that disease of the two hemispheres occurs in a nearly equal proportion, for out of 110 cases, there were 58 in which the lesion was in the right, and 52 where it was in the left hemisphere.

The centres of innervation are doubly arranged for all the different kinds of work which it falls to the lot of man to do; nevertheless most people are left-brained and right-handed. For most kinds of finer work, such as writing, drawing, &c., only the left hemisphere is trained, even by persons who are in other respects left-handed; and only for a few kinds of work both hemispheres have to be educated. This is chiefly the case with musicians. Pianists have to train both hemispheres similarly, while violinists and violoncello-players have to educate them dissimilarly. That, as a rule, the left hemisphere is trained in preference to the right, for language is, according to Dr. William Ogle, due to the following circumstances: greater weight and specific gravity of left hemisphere; greater development of convolutions in left frontal parts; earlier fœtal development of left hemisphere, and greater supply of blood to it; and, finally, greater width of the left carotid artery than the right.

Aphasia may in children and young persons be only temporary, as they are able to train the right hemisphere after loss of the left; just as persons, who suffer from scriveners' palsy in the right hand, may train the right hemisphere for writing with the left hand.

Dr. Ogle has done good service by dividing aphasia into two classes: viz., amnemonic and atactic aphasia. In the *amnemonic* variety there is difficulty of remembering the spoken or written word, while in the *atactic* variety there is loss of motor co-ordination of words. In the former variety the idea is there, but the idea does not suggest the proper symbol; and either no word or a different one, with a different meaning, is forthcoming. In this latter case it is curious to notice that the grammatical form is observed—substantives being substituted

for substantives, verbs for verbs, proper names for proper names, &c. In the atactic variety, on the other hand, the patient is unable to say the words which are suggested by the memory, for want of co-ordinating power over the muscles of articulation. Patients of this class are generally either entirely deprived of speech, or say only 'yes' or 'no' or some other monosyllable. The two varieties may occur together or separately. It is, therefore, probable that each of these faculties has its separate centre, capable of a separate lesion, but that the centres lie so near one another that the same lesion is likely to injure both.

An interesting case of atactic aphasia is that which M. Lordat,[1] Professor of Medicine at the University of Montpellier, has described as having occurred in his own person. He suddenly lost the faculty of speaking and reading after a fever, yet was able to think and to go mentally through the whole course of his lectures. He heard the spoken words, but they remained unintelligible. He had, however, only lost his syntax and not his alphabet; for though unable to read written words, he could read letters. This state of things had been going on for several months, when suddenly one day, while he was in his library and glanced over the bookshelves, the words 'Hippocratis opera' appeared to him. Tears rushed from his eyes, for he saw that he was better, and he ultimately regained the power of speaking and reading. Yet he did not entirely recover, for, from having been an excellent *extempore* speaker, he was afterwards obliged to read his lectures from a copy.

The same considerations which apply to the spoken language also apply to written words. Dr. Ogle has called the loss of the faculty of intelligent writing *agraphia*, and again distinguished the amnemonic and atactic variety. In the former the patient can write letters and words, but without rhyme or reason, so that the letters do not convey any meaning; while in the latter variety the power of writing separate letters is lost. Sometimes in the amnemonic variety there is still power to write a few words properly. A lady aged sixty, who was some years ago under my care, and who had lost her speech five years before, could with difficulty write her own and her husband's name, but not

[1] 'Analyse de la Parole.' Montpellier, 1843.

quite grammatically, as instead of 'Jackson' she wrote 'Jacton,' instead of 'Rectory,' 'Bignoral,' and instead of '1866' '1665.' She had had right hemiplegia, from which however she had recovered, and there was no motor paralysis at the time. She could dress and feed herself, do her hair, thread her needle and sew, make pies and puddings, and walk four or five miles without fatigue; yet she could only write by holding the pen in the left hand and then guiding it with the right. She could say 'yes' and 'no,' and occasionally said 'indeed' and 'thank you.' But sometimes she would say 'yes' when she meant 'no,' and *vice versâ*; and she then corrected herself by pantomime, as when she nodded her head, she always meant 'yes,' and when she shook it, she always meant 'no.'

Aphasia and agraphia generally occur together, but they are occasionally separate, which points to the existence of distinct cerebral centres for the two faculties, which are in close proximity to each other.

The loss of pantomimic expression (*amimia*) is much rarer than aphasia and agraphia, and points to a more extensive lesion than the latter.

For all these conditions we find numerous analogies in persons whose brain is on the whole working physiologically. Forgetfulness of words, more especially names and dates, is common in the aged, although not amounting to amnemonic aphasia. A well-known university professor, who forgets his own name, and on being asked for it by a stranger to whom he had been courteous, buttonholes a passing friend, and exclaims in a distressed tone: 'For heaven's sake, tell me my name!'—is an instance of this. In the same case there was sometimes difficulty in properly appreciating written words, of which the following is an instance. Professor —— had for some time been in the habit, when going out, of hanging a card over his library door, on which he had written the words: 'Professor —— is not at home.' One evening, when returning home, he stopped at the door, looked at the card, said, 'How very annoying that that man is not at home!' and went away again.

Some persons who have entirely lost their language are still able to play chess, backgammon, and whist; and they have been observed to cheat at cards with some ingenuity. They may also

be sharp in business matters. Musicians who suffer from aphasia and agraphia of written words may still be able to compose music, and write it down in notes. The intellect and speech do not run in identical grooves; and if aphasia is connected with impairment of the mental faculties, the latter is not owing to the former, but both are caused by a more extensive cerebral lesion.

There is another variety of aphasia, which Kussmaul has called '*verbal deafness and blindness*,' in which the patient is still able to speak and write words, but does not understand anything that is said to him, although the hearing is quite good; nor anything that is written, although the sight is good. The patient who suffers from verbal deafness is sometimes believed to be deaf or insane, because he speaks words in a senseless connection, and does not seem to understand what is said; but his thoughts and hearing are perfectly correct, and he is in the position of an European who should suddenly find himself in the midst of South Sea Islanders, whom he would hear talk, but could not understand, nor would he be understood when talking to them in his own language.

In some cases there is a perfect chorea or delirium of words, which may be called *paraphasia*. Dr. Osborne[1] has recorded a case in which the intellect of the patient was unimpaired, and who could express himself well by writing, understood everything that was said to him and what was given him to read; but the connection between ideas and spoken words was broken, so that when he attempted to talk, or to read printed matter, an unintelligible jargon was the result. For instance, he read a sentence from the bye-laws of the College of Physicians, viz., 'It shall be in the power of the college to examine or not examine any licentiate, previously to his admission to a fellowship, as they shall think fit,' as follows:—

'*An the be what in the temother of the trothotodoo to majorum or that emidrate ein einkrastrai mestreit to ketra totombreidei to ra fromtreido asthat kekritest.*'

The same passage was presented to him a few days afterwards, when he read it as follows:—

'*Be mather be in the kondreit of the compestret to samtreis*

[1] 'Dublin Quarterly Journal', November 1833, p. 157.

amtreit emtreido am temtreido mestreiterso to his eftreido tum bried roderiso of deid daf drit des trest.'

Dr. Osborne observes in the above jargon 'several syllables of frequent occurrence in the German language, which had probably made a strong impression on the memory of the patient,' who had been a good linguist; but to this I must demur, the words given being particularly unlike German; and it seems much more likely that they were, if anything, reminiscences of old native Irish.

In the same manner we might speak of *paragraphia* and *paramimia*, in cases where the patient is able to write or gesticulate, but does so in a way which is wrong or unintelligible.

There is a kind of *functional aphasia*, in which connection between the cortex and the tongue is severed, either by mental emotions, more particularly fright, or in consequence of fevers, especially typhoid, but also scarlet fever, measles, erysipelas, &c.; and which appears also to be congenital. Herodotus relates that the son of Crœsus was dumb; but that when after the capture of Sardes he saw a Persian running, with sword drawn, to kill his father, he exclaimed: 'Man, do not kill Crœsus!' and he afterwards retained the use of language. Kussmaul mentions the case of an hysterical woman who lost her speech on her wedding-day, and only regained it when she saw a church on fire. She then screamed, 'Fire!' and spoke as well as ever after that.

2. *General Paralysis of the Insane* (*Dementia Paralytica*, *Folie Paralytique*).

This disease, which has been for the last decennia a favourite subject of study for alienists and neuro-pathologists, consists essentially of a diffuse chronic interstitial inflammation of the brain and spinal cord, which leads in time to destruction of ganglion cells and wasting of the nervous centres. It may aptly be compared to the atrophic form of Bright's disease of the kidneys. Its progress is generally slow, but occasionally acute. In the latter case symptoms of inflammation predominate: the brain appears swollen, the convolutions bulky, the fissures small, the cortex thickened, and on section hyperæmia and multiple softening is discovered. The coats of the arterioles

and capillaries are studded with nuclei, and distended by white corpuscles. In the great majority of cases, however, the disease has a more protracted course, and leads to atrophy, as the ultimate result of the inflammation. The dura mater adheres firmly to the skull, and may show thickened and opaque osteomata. The pia mater is œdematous, and a quantity of serum is found over the parietal and occipital lobes of the brain; the membrane often appears white or yellow, and is thickened chiefly at the sides of the longitudinal fissure, and in the neighbourhood of the large bloodvessels. The pia generally adheres to the surface of the brain, and on attempting to separate it, the cineritious substance is torn. The convolutions, more especially those of the anterior lobes, are wasted, and changed into an inert mass. The ganglionic cells are completely altered in form and colour, and have undergone retrogressive degeneration. . Their place is taken by amylaceous bodies, and a quantity of new connective tissue, which binds the structure together and hardens it. The bloodvessels of the brain sometimes undergo a calcareous change, standing out like bristles; they appear tortuous and varicose. Dr. Lockhart Clarke has also found widening of the peri-vascular canals (i.e. the sheaths of the cerebral bloodvessels), from wasting of the brain-tissue, and granules of hæmatoidine in the sheaths and the vessels. The ventricles of the brain are dilated, and the ependyma is covered with small or large rough granulations; the medullary matter is softened, and the surface of the central ganglia puckered. The weight of the brain is much diminished, and often amounts only to thirty ounces. In the spinal cord the signs of sclerosis of the posterior columns are met with; viz., wasting of nerve-fibres, proliferation of connective tissue, and formation of oil-globules and amyloid corpuscles: or those of granular myelitis (Westphal), viz., enormous masses of fat-globules, chiefly in the neighbourhood of the bloodvessels of the lateral columns, with thickening of the septa of the cord. Similar changes have been discovered in the cerebral nerves and the posterior roots of the spinal nerves. The optic discs are generally wasted.

General paralysis of the insane affects chiefly males, the proportion being of about seven men to one woman; and the age at which it proves most fatal is between thirty and forty-

five. The greater prevalence of the disease in the male sex is probably owing to the fact that males are more exposed to injurious influences, and drink harder, than women. Hard work, combined with anxiety and excesses in alcohol and sexual indulgence, seem to predispose to the malady; but injury to the head, affecting the cineritious substance, is no doubt frequently the real cause. The disease may also become developed in persons saturated with the syphilitic poison, and in convalescents from acute diseases. It is more frequent amongst the lower orders than the higher classes, for many more cases of it are seen in public than in private asylums. Whether the affection is hereditary or not, remains as yet an open question.

As a rule the brain is first affected, but in some cases symptoms pointing to disease of the spinal cord, such as progressive locomotor ataxy, or paraplegia, precede the cerebral symptoms. The disease then gradually creeps upwards through the pyramids into the crura cerebri, the corona radiata, and ultimately into the cortical substance of the hemispheres.

The first symptoms are those of mental excitement. There is considerable irritability of temper; the memory is impaired, chiefly for recent occurrences and names; and vertigo and headache are complained of. Little oddities in the patient's behaviour also begin to attract attention; he loses his self-control and reasoning powers; but the most important symptom at this stage is a peculiar tremulous or quivering motion about the lips and facial muscles generally. The speech now becomes altered; it assumes a nasal twang; articulation is difficult, and sometimes there is actual stammering, which, however, may be overcome by an effort of the will. These symptoms point to an affection of the medulla oblongata, or the cerebral ganglia of the hemispheres; but in other cases there is an embarrassment of language, which partakes more of the nature of aphasia, and must be ascribed to degeneration of the cineritious structure of the convolutions. In such cases articulation may still be quite perfect, and there is no apparent loss of power in the lips, tongue, and palate. Temporary fits of complete aphasia may also occur under these circumstances. Sometimes the pitch of the voice is altered; in most cases it is lowered. Differences in the size of the pupils are also noticed, one being

larger than the other, which is a most valuable sign to the examiner of a proposal for life insurance. As a rule the pupils are narrow.

This stage of the disease may last for several months, after which other important symptoms make their appearance. There is a decided want of power, but as yet no paralysis. The speech is thick, like that of a drunken man, and sometimes quite unintelligible; the saliva runs away; the expression is vacant and silly; the tongue is put out with difficulty, and appears tremulous when protruded. The patient is apt to smack the tongue and grind the teeth. The walk is often grotesque; the patient sets off at a tremendous pace, then suddenly stops, and will not go any further; or he stops every minute, and wants much persuasion to proceed. There is tremor in the hand; he can no longer dress or feed himself properly. There is inability to write, or at least great clumsiness in guiding the pen. The character of the handwriting is altered, and mistakes in spelling are frequent. At the same time the memory fails more largely; the patient becomes unable to manage his business; or, if he is left to attend to it, makes fatal mistakes which may ruin himself and his family. He loses all ideas of the value of money, and signs away large sums for trumpery things. If unmarried, he will contract an absurd alliance. In the married the affections are weakened; he does no longer care for his family. If contradicted about a slight matter, he is apt to fly into a terrible rage. At the same time there are delusions, which have a decidedly ambitious and exalted character. The patient often becomes an emperor or a king, lives in a palace, and has an annual revenue of a million sterling; he has ten thousand horses in his stables, and feels perfectly happy in this magnificence. He shows an incapacity for dwelling on painful or troublesome subjects. In other cases, again, the mind seems a perfect blank, or perverse impulses are developed. Kleptomania is one of these, and such patients have been sent to prison with hard labour instead of to an asylum. Attempts at rape and homicidal mania also occur, and it is therefore generally advisable to place the patient under restraint. Depression sometimes alternates with exaltation, and although the loss of physical power is great, yet in sudden attacks of maniacal excitement a prodigious force is

sometimes developed. This oscillation between depression and irritation is generally accompanied with analogous changes in the pulse, which is very slow, viz. from 40 to 50 beats in the former condition, while it may rapidly rise to 100 or 120 as soon as exacerbation becomes established.

In the last stage of the disease there is paralysis of motion and imbecility. It sometimes commences with a fit of apoplexy, followed by hemiplegia. The latter may improve as time goes on, but the intellect becomes progressively more clouded and physical decay more marked. The patient now leads a vegetative existence; he sits in a chair, or lies in bed, callous to anything which may go on around him. Sometimes he shows the symptoms of complete paralysis of the portio dura on one side; the body may be anæsthetic, so that surgical operations are undergone without flinching, and self-mutilation is practised without evidence of pain. Epileptiform seizures occur at this time in all their various forms, from the slightest attack of 'petit mal' to the most violent fit of convulsions, and the patient sometimes dies in a fit two or three years after the first appearance of the symptoms, or he becomes bed-ridden, affected with paralysis of the sphincters, and is ultimately carried off by decubitus, pneumonia, or acute tuberculosis. In such cases there may be great wasting of flesh, or an excessive development of fat.

In exceptional cases the disease lasts much longer, and ends in recovery; while, on the other hand, it may run an exceedingly rapid course, and end fatally in a few months.

3. *Paralysis agitans* (*Shaking Palsy, Parkinson's*[1] *Disease*).

This disease, which has frequently been confounded with senile tremor, chorea, and multiple sclerosis of the cerebro-spinal centre, is characterised by two principal symptoms, viz. tremor and paralysis of voluntary muscles, both being decidedly progressive in their course. The tremor precedes the paralysis, and generally commences in a single limb, mostly one of the upper extremities; but where a powerful cause—such as terror or a great shock—acts suddenly, it may at once affect all four

[1] 'Essay on the Shaking Palsy.' London, 1817.

extremities. I have seen this in the case of a woman aged fifty-nine, whose husband had been accidentally killed before her eyes.

The tremor varies considerably in degree, from hardly perceptible vibrations to violent clonic convulsions. Sometimes it is so slight that it escapes the eye except on the most careful observation, but reveals itself at once to the sense of touch. It also affects the neck, and more particularly the sterno-mastoid and trapezius muscles, by which the head is moved forward and laterally. In cases of long standing this peculiarity is sometimes neutralised by paralysis of the long extensors of the spine, which is followed by tonic contraction of the sterno-cleido-mastoid muscles. The head then falls forwards, the chin rests on the sternum, and sometimes great force is necessary to pull the head back so as to give it for a time its proper position.

The tremor becomes less in the horizontal position, and ceases completely during sleep. Even those patients whose limbs are most violently agitated throughout the day, sleep as quietly as children. The shaking re-commences, however, almost immediately after awakening; and becomes as bad as ever on getting up. It is much increased by excitement and by efforts of the will to stop it; but it can be arrested for a minute—where it occurs in the upper extremity—by knocking the hand forcibly on the knee or a table, and in the leg, by stamping the foot on the ground. It also becomes less when the patient makes a voluntary movement of whatever kind.

Paralysis only appears after the tremor has lasted for a more or less considerable time. There is sometimes only a slowness in carrying out intended movements, and great muscular fatigue after insignificant efforts; while in other cases there is nearly complete paralysis, excepting only the muscles of the eyes. As a rule the palsy affects chiefly the extensor muscles, and is connected with greater contractions of the antagonistic flexors than are found in other forms of paralysis. The body therefore eventually becomes quite deformed. Not only does the head droop, but the entire figure is bent forwards; the arms are rigid; the fingers half flexed, as if for writing; the knees are so closely approached to one another that it is difficult to separate them; and the foot assumes the position of

pes equinus. This extreme muscular rigidity makes the patients peculiarly helpless, and they require powerful attendants to minister to their wants.

A singular feature of shaking palsy is the tendency to forced movements. Some patients have the impulse to run forwards, and find it impossible to check it; and they often fall down, as they run in an awkward manner. One of my patients could only move about in the following way:—he clasped with both hands the hands of his attendant, and then commenced running briskly backwards while the attendant followed him running forwards, and thus kept the invalid's balance. Some patients can walk but cannot stand. In one remarkable case which I had the opportunity of observing for two years at the Hospital, the patient, when placed upright in the centre of a room, would at once reel backwards until he found a support for his back—such as a table or the wall; and he could then stand for a long time. He had a difficulty in getting started for walking; but when he had once commenced, he could walk several miles without stopping. When he was obliged to stop, he would reel back directly. On crossing a street he had therefore to walk round and round if the road was not clear, as standing still was out of the question. He had no difficulty in going downstairs, but found it impossible to go uphill. The most singular feature of this case, however, was that the patient only had the use of his arms and hands while walking. When standing and resting his back against the wall, his hands were utterly paralysed; but as soon as he set off walking through the room, he could move his arms in all directions. Thus, it was while walking that he dressed and undressed himself, being too poor to pay for an attendant; and he even took his meals while promenading about his room, unless there happened to be somebody charitable enough to feed him. I unfortunately lost sight of this case before its fatal termination, but I should, in a post-mortem examination, have expected to find disease of the posterior part of the middle lobe of the cerebellum.

In spite of the incessant muscular action, there is no increase of temperature on the surface. The patient, however, has a feeling of increased heat, particularly when the shaking is violent. The faradic and galvanic excitability of the muscles

is generally normal, but where there is a high degree of paralysis and contraction it appears diminished. Sensibility only becomes affected in the later stages of the complaint, when there are sensations of pins and needles, numbness, and a degree of anæsthesia in the arms and legs. The speech is often drawling and indistinct; the patient, instead of 'yes' says 'yeeeeeees' —sometimes spreading one such word over nearly a minute. The mental faculties also become dulled; there is headache, giddiness, depression, and not rarely delirium in the small hours of the morning. There may be profuse perspiration, which I have occasionally found to have a most villanous smell, even in persons who were well attended to and quite cleanly in their habits.

The course of the disease is protracted over a number of years. It generally occurs after fifty years of age. Most of my patients were between sixty and seventy, but I have seen one who was only forty. The chief causes are anxiety, losses in business, hard work and bad living, and lastly injury. It is much more frequent in males than in females. In my practice the proportion has been 10 males to 1 female, but this does not appear to be the general rate; for amongst 69 fatal cases which occurred in 1874 in England and Wales, there were 42 males and 27 females. Death takes place from exhaustion of the nervous force, and is often preceded by decubitus, pneumonia, and other acute diseases.

No peculiar anatomical lesion appears to be invariably connected with shaking palsy. Some observers have found sclerosis of the corpora quadrigemina, pons, medulla oblongata, and spinal cord, and wasting of the thalamus opticus and cornu Ammonis; but in many cases the results have been negative.

4. *Labio-glosso-pharyngeal Paralysis, progressive bulbar Paralysis, Paralysie labio-glosso-laryngée progressive, Duchenne's Disease.*

We have seen in the first chapter that the medulla oblongata is the co-ordinating centre of the movements of *deglutition*, for which it associates the muscles of the lips, tongue, palate, and pharynx, and of articulate *speech*; the integrity of pronunciation of letters being bound to the integrity of the motor

nuclei of the hypoglossus, pneumogastric, and spinal accessory nerves. A most interesting pathological illustration of these physiological conditions is given by the disease which we have now to consider.

Progressive bulbar paralysis was first described in 1860 by Duchenne, who believed it to be a muscular disease; but Trousseau, who had soon afterwards the opportunity of making several autopsies, was led to the conclusion that it was rather a disease of the nerves than the muscles, and that it was owing to atrophy of the nerve-roots. Since then a number of post-mortem examinations have been made by Charcot, Joffroy, Kussmaul and others; and the microscopic examination of the medulla oblongata, according to Dr. Lockhart Clarke's method, has yielded most satisfactory results, which in a very great measure explain the symptoms of the disease.

Examination of the medulla by simple inspection teaches us little or nothing; but microscopic specimens which have been hardened in chromic acid or bichromate of potash, coloured by carmine and rendered transparent by glycerine, show marked changes in the nuclei of the motor nerves. The application of chromic acid allows us to distinguish healthy and diseased parts by the naked eye, for the former then assume a dark-yellow colour, while the latter remain light. On the other hand, carmine colours the diseased parts more deeply, but the healthy parts, which have already taken up the chromic acid, absorb carmine less easily. Another peculiarity is, that carmine colours the ganglion cells, their processes, and the neuroglia, but not the nerve-fibres, which are therefore plainly perceptible. By using these tests it is discovered that there is degeneration of the ganglion cells in the motor nuclei of the rhomboid fossa, which generally assumes the form of yellow pigmentary atrophy. The ganglionic cells appear dark yellow or reddish yellow, greatly shrunk in size, and with their processes indistinct or absent. The nucleus of the cell perishes, and its place is taken by brown granules which appear in crowds. Ultimately, nothing remains of the cell except yellow granules, which are seen singly or heaped together.

Of the nuclei of nerves in the rhomboid fossa, that of the hypoglossus is most constantly affected. In a case which Charcot

observed together with Joffroy, he compared one of Dr. Lockhart Clarke's original preparations of a normal hypoglossus-nucleus with that of the diseased specimen, and found that the number of ganglion cells in the wasted nucleus amounted to only about $\frac{1}{10}$th or $\frac{1}{12}$th part of those contained in the normal preparation. The nuclei of the accessory and pneumogastric nerves are also most generally affected, while those of the portio dura and the motor portion of the fifth are more rarely diseased, and the sentient nucleus of the fifth, the auditory, and glosso-pharyngeal nerve are found normal.

Another anatomical change which has been discovered is chronic sclerosing myelitis. This was shown by finding numberless oil-globules in the medullary matter; the neuroglia had become granular or fibrous, and appeared proliferated by showing larger spaces between the several nerve-fibres. The blood-vessels were thickened and surrounded by a large sheath of connective tissue, in which oil-globules and shining disc-like corpuscles could be seen. The nerve-fibres appeared much reduced in number and size, their contents were in a state of granular and fatty degeneration, and the cylinder axis was either thickened or wasted and entirely gone. This myelitis was chiefly found in the rhomboid fossa and the anterior pyramids, less in the corpora olivaria, and not at all in the restiform tracts.

That the corpora olivaria should only slightly suffer in this disease speaks strongly against Schröder van der Kolck's theory of these bodies being entrusted with the function of regulating articulation. Jaccoud has accepted this theory, but Dr. Lockhart Clarke has shown that parrots—which have great facility in imitating articulate sounds—have no olivary bodies at all, and that seals—who never articulate—are provided with enormous corpora olivaria. These facts suffice to render Van der Kolck's theory untenable.

Although therefore a definite lesion has been discovered which accounts for the symptoms observed in patients suffering from progressive bulbar paralysis, it is nevertheless a singular fact that in some cases the wasting of ganglion cells has not been proportionate to the wasting of the muscles which are under their influence. Thus there has been great wasting of

the nucleus of the hypoglossus, where there was only a slight degree of atrophy of the tongue. Brown-Séquard and Charcot have attempted to explain this by assuming that there is a difference between simple atrophy and wasting accompanied with irritation, and that the former only causes paralysis while the latter will produce paralysis and atrophy combined. On the other hand, Duchenne and Joffroy are of opinion that the ganglion cells in the motor nuclei are partly motor and partly trophic. Wasting of the motor nuclei would, according to them, cause paralysis; while atrophy of the trophic structures would lead to atrophy as well. No proofs have as yet been given in support of either theory.

Progressive bulbar paralysis seems to occur more in males than in females. It is rare before thirty years of age; appears to follow cold, mental anxiety, and injuries to the head; and is sometimes complicated with progressive muscular atrophy. It is not really a special disease, but a complex of symptoms owing to a peculiar degeneration; and it shows in this respect the greatest analogy to progressive locomotor ataxy, chronic diffuse myelitis, multiple sclerosis of the nervous centres, and secondary degeneration of the lateral columns of the spinal cord after an attack of cerebral hæmorrhage. If it generally kills more rapidly than the diseases which I have just mentioned, this is owing to the affected organ being more essential to life (*nœud-vital*, p. 37). While therefore ataxy will sometimes be protracted over twenty years, progressive bulbar paralysis rarely lasts longer than two or three years and oftentimes not so long, since it kills its victims either by suffocation from morsels of food arriving in the air-passages, or by inability to expectorate mucus, or finally by apnœa and syncope.

The commencement of the disease is generally insidious. The patient finds that eating and talking fatigue him; the tongue feels heavy, and deglutition is uncomfortable. Sometimes he also complains of headache, pain at the nape of the neck, and a feeling of constriction in the throat and the chest. An early symptom may be excessive salivation, which cannot be explained by the mouth being generally kept open, but is most probably a neuro-lytic phenomenon; for Claude Bernard has found extreme salivation in animals in which he had divided

the tympanico-lingual nerve, which is a branch of the portio dura.

After these symptoms have lasted a variable time, a sudden change for the worse takes place in the patient's condition, which seems sometimes to be owing to a severe cold, but may also occur without a perceptible cause.

Symptoms of impaired articulation (*alalia*, *anarthria*), are now prominent. There is not only general difficulty in speaking, which becomes slower and laboured, but also loss of certain letters, which depends in its essential features upon the course which the destruction takes in the motor nuclei, as it varies in proportion to the affection of the lips, tongue and palate. Where the *lips* suffer principally, the consonants *b*, *p*, *f*, *m*, and *v* become difficult or impossible; just as is found in persons suffering from hare-lip which has not been operated upon. The vowels which chiefly suffer are *o* and *u*; afterwards *e* and *a*. Helmholtz has shown that we pronounce the vowels by effecting certain changes in the shape of the cavity of the mouth, more especially by the action of the lips; and that the vocal cords by their vibrations only serve to alter the pitch in which the vowels are spoken. In a case of total occlusion of the larynx, in which laryngotomy had been performed, the patient could neither talk nor whisper; nevertheless she contrived to lisp by alternately increasing and reducing the column of air in the cavity of the mouth.

On account of this paralysis of the lips, the patient is unable to whistle or to blow out a candle. The respiratory portion of the facial nerve is the only one affected in progressive bulbar paralysis, causing loss of power in the muscles of the mouth and nose, while the orbicularis palpebrarum, and other muscles which serve for physiognomical expression, remain in their normal condition. By paralysis of the respiratory muscles of the mouth and nose, the mouth appears wider, or quite open; the naso-labial sulcus is deeper, and the face assumes a lachrymose expression. On examining the affected muscles by galvanism and faradism, it is seen that their contractile power becomes diminished as atrophy sets in; but as long as muscular fibres are still present, these will respond, though sluggishly, to both kinds of current. The reflex excitability of these muscles

is likewise diminished or lost in the further progress of the disease.

Where there is difficulty in raising the *tongue* to the roof of the mouth, the vowel *e* is lost, and the consonants *r*, *sh*, *s*, *l*, *k*, *g*, *t*, *d*, and *n*. The same difficulties are observed where there is congenital or acquired defect of the tongue, for instance, after an operation for cancer; in congenital and acquired hypertrophy of the organ; where the frenulum is too short or too firm; in tumours; and also where the movements of the member are impeded by contraction and anchylosis of the jaws.

Paralysis of the *soft palate* produces a nasal twang, and impedes the pronunciation of the letters *b* and *p*, because the current of air which goes through the mouth, is not powerful enough to overcome the resistance of the lips. That such is really the case is shown by the fact that such patients are still able to pronounce *b* and *p* by closing their nostrils, whereby the force of the blast is increased. The nasal twang is owing to the soft palate being unable to close up the posterior opening of the nose during the pronunciation of the vowels and of all consonants, excepting *m*, *n*, and *ng*. Such a twang is therefore the same as is observed in diphtheritic paralysis of the soft palate, in syphilitic ulceration, and in congenital fissure of hard and soft palate. Hypertrophied tonsils, polypus, and other diseases cause similar symptoms. *B* and *p* are sometimes pronounced as *m* and *f*.

When all letters are lost, there is *alalia*, and this is invariably combined with *aphonia*, from paralysis of the vocal cords. The patients are ultimately only able to grunt, and an examination by the laryngeal mirror shows that the vocal cords have ceased to vibrate. At the same time expiration and expectoration become difficult. The patient finds himself unable to clear his nose and throat, or to cough at will, although he may still cough when foreign bodies, such as particles of food, get into the larynx. When this stage has been reached, dyspnœa and syncope from paralysis of the pneumogastric nerve are to be feared.

Another function which suffers and becomes ultimately annihilated in progressive bulbar paralysis, is that of *deglutition*; and the distress caused by this must have been witnessed to be

realised. Where the mouth cannot be properly closed, the food which is taken is apt to fall out again. The tongue has no longer any power in assisting the movements of mastication and the formation of the bolus; the morsel therefore remains lying between the teeth and cheeks, until the patient helps its progress downwards by pushing it on with his fingers and throwing the head backwards. Where the pharynx becomes paralysed, large pieces of food are apt to remain in it, and may cause suffocation by entering the air-passages. In some cases liquids, and in others a solid morsel, is more easily swallowed, according to the portion of the throat which is most affected. The tongue and the muscles of the pharynx ultimately become wasted, flabby, and tremulous; they show fibrillary twitches which are characteristic of muscular atrophy, and yet remain sensitive to Faradisation until the entire muscular structure is destroyed.

Insufficient nutrition does not only constitute one of the most painful features of this disease, but also causes great systemic debility, from inanition. The patient is therefore soon confined to his bed or couch, and finds it very difficult to move about. In a number of cases the cervical portion of the cord becomes involved, causing the symptoms of progressive muscular atrophy, at first chiefly in the thumb and the other fingers, and afterwards affecting the whole of the upper and lower extremities. Respiration is now imperfect; the skin is generally covered with clammy perspiration; and paralysis of the sphincters and delirium may set in towards the end. But generally the mind remains perfectly clear; the patient spends his time in brooding over his terrible condition, and longs for the final deliverance which alone can put an end to his unbearable troubles.

It is not only pigmentary degeneration and sclerosis of the motor nuclei of the medulla which lead to difficulty or impossibility of articulation, but any other morbid process by which these nuclei are disabled will cause a similar effect. Such are hæmorrhage, inflammatory or embolic softening, abscess and tumour of the medulla; as also injury or disease of the skull and periosteum, pressure of tumours of the cerebellum, and propagation of any acute or chronic disease from the pons or spinal

cord to the fourth ventricle. On the other hand, the medulla is sometimes found diseased without there having been any symptoms of alalia or anarthria during life; and this is explained by the fact that the medulla is a complex organ, including a large number of different centres and conducting fibres in different situations. The symptoms which are observed during life depend strictly upon the portion of the medulla which is diseased.

Difficult articulation is also met with in disseminated scleroris of the cerebro-spinal centre. In sclerosis of the cord alone, the speech is not affected; but where the medulla, and the central ganglia of the hemispheres suffer, dysarthria is generally the result. This disease does not however produce aphasia, as it spares the cortical centres of the hemispheres.

Drs. Silver and Cheadle have described cases in which symptoms of bulbar paralysis were owing to syphilitic infection of the system, and cured by iodide of potassium. These cases, however, do not quite fit into the frame of Duchenne's and Trousseau's description of 'progressive labio-glosso-laryngeal paralysis.' The dysphagia and dysarthria of hysterical women likewise belongs to an entirely different department of pathology.

5. *Diphtheritic Paralysis.*

Palsies after certain acute diseases, such as typhoid fever, cholera, small-pox, etc., have been known for a considerable time; but the connection which exists between diphtheria of the fauces and a peculiar form of paralysis was only pointed out by Bretonneau, Trousseau, and Lasègue. That this should have escaped the attention of previous observers, is probably owing to the circumstance that in most cases the paralysis does not immediately follow upon diphtheria, but that a considerable interval elapses between the primary and secondary affection. This interval often amounts to two or three weeks, but it may be as many months.

There are two forms of this palsy, one of which is attended at once with severe constitutional disturbance; there being nephritis, dropsy, œdema of the lungs, general paralysis, uræmic convulsions and delirium; and death takes place in a few

days. But in most cases the affection is milder and generally ends in recovery, unless the patient should accidentally choke by a morsel of food finding its way into the air-passages.

This latter form is distinguished by the peculiar succession of paralytic symptoms. The first part to suffer is the *soft palate*, which is shown by the patient speaking with a nasal twang; and articulation, more especially of the guttural sounds, becomes difficult or impossible. On inspecting the soft palate, it appears flabby and drooping; and when the tongue is depressed by a spatula, the palate is seen to remain immovable when irritated, instead of going up and down as in health. It may be touched, pricked, and even cauterised, without showing signs of reflex excitability. If the patient be made to expire forcibly, the palate remains motionless. Sometimes one half of it is more affected than the other half. In consequence of this the deglutition of liquids suffers, and they may be returned through the nose. Where the pharyngeal muscles are likewise paralysed, deglutition of solids is impaired; the morsel of food is apt to lodge in the folds of the pharynx, and may cause suffocation by entering the glottis.

The tongue and larynx are rarely affected, but paralysis of the motor nerves of the eyes is very common. The ciliary nerves which are supplied by the lenticular ganglion are more especially liable to it, causing diminished or lost power of accommodation, so that the patient cannot read or see small objects, but can see well in the distance. There may also be ptosis, strabismus and diplopia, owing to paralysis of the third nerve; but what used to be called 'diphtheritic amaurosis' does not exist, as there is no optic neuritis, and the ophthalmoscope shows the fundus of the eye to be perfectly healthy.

Paralysis of motion also affects the extremities. The patient experiences great difficulty in standing and walking. The dynamometer shows diminished grasping power of the hands. The neck is also affected, so that the head may tumble about on the pillow from one side to the other, the patient being unable to fix it. Affection of the diaphragm is shown by dyspnœa and accelerated respiration; while the very slow pulse, which is generally met with, shows loss of power in the sympathetic ganglia of the heart, with preponderance of the inhibitory

action of the pneumogastic nerve. The pulse falls often as low as 42, and even less.

Diphtheritic paralysis resembles that which is observed after other acute diseases and in hysterical women by a peculiar tendency to shift its place; it will leave one limb to go to another, and then spread to the rectum and bladder, causing incontinence or retention. There may also be loss of the virile power.

Sensibility suffers frequently. The gums may be so anæsthetic that the teeth do not feel the contact with the food. There are 'pins and needles' in the fingers; the sense of touch is dull, and the anæsthesia may be complete in the hands and arms, but rarely spreads over the whole body. Nevertheless the lower extremities do not escape: for the patient has the peculiar sensation as if he was walking on cotton; he cannot feel the ground properly, and must have the assistance of the eyes in walking.

The degree of the palsy is not proportionate to the severity of the primary affection; for it often comes on after a mild attack of pharyngeal diphtheria. Nor can the paralysis of the soft palate be ascribed to the local influence of the diphtheritic poison, because it may only come on long after the poison has been eliminated from the pharynx; and it has also been observed when the diphtheria did not affect the fauces, but the skin.

Von Graefe attributed the origin of diphtheritic paralysis to an affection of the sympathetic nerve; and Dr. H. Jackson seems inclined to coincide in this opinion. It is however more probable that it is owing to migrating neuritis. Buhl found inflammatory proliferation of the nuclei of the connective tissue of the sheaths of the peripheral nerves and the anterior and posterior spinal nerve-roots. The inflammatory effusion may be absorbed, and in such cases recovery takes place, or the proliferated connective tissue shrinks and becomes sclerosed, when pressure on and constriction of the nerves will cause more or less permanent paralysis and anæsthesia. In such cases there is generally atrophy of the paralysed muscles, with more or less complete loss of their faradic and galvanic sensibility. Oertel has also found hæmorrhage in the spinal meninges, and great proliferation of nuclei in the nerve-sheaths and the grey substance of the centre

of the cord. The albuminuria which is so frequently present in these cases does not seem to have any influence upon the degree or extent of the paralysis.

Peripheral paralysis from injury to the nerves, infantile paralysis, palsy occurring after acute diseases, such as typhoid fever, small-pox, scarlatina, pneumonia, erysipelas, etc.; rheumatic, reflex, and hysterical paralysis, as well as lead palsy, will be discussed when the affections of the peripheral nerves come under consideration.

CHAPTER V.

CEPHALITIS.

CEPHALITIS, by which name the various inflammatory conditions of the nervous centres are comprehended, caused the following mortality during six periods of five years each, in England and Wales :—

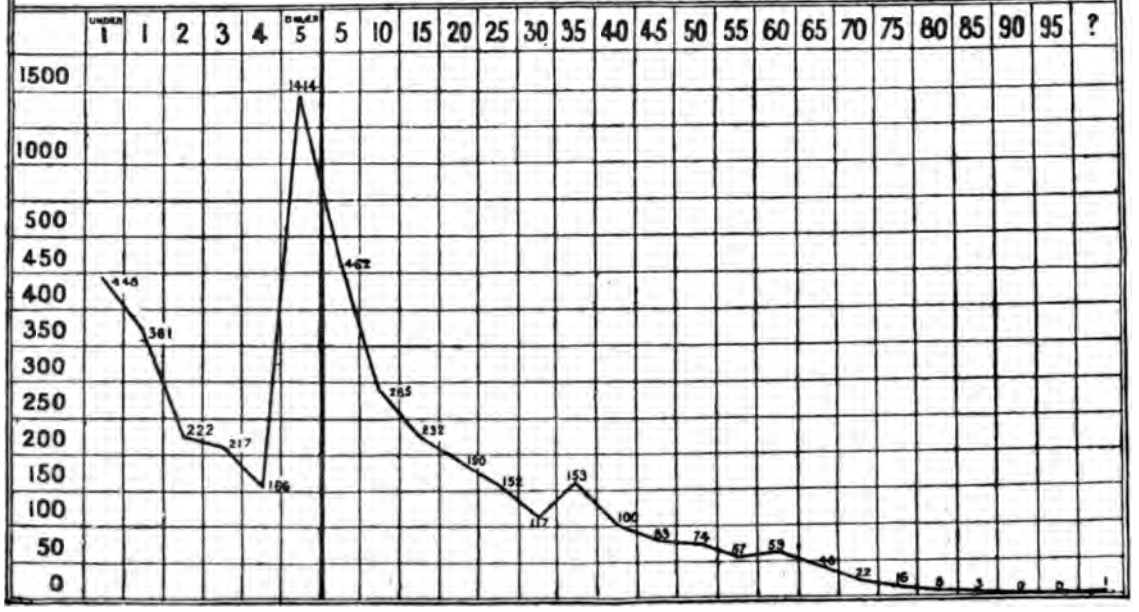
Periods of Five Years	Deaths from Cephalitis	Percentage of Nervous Diseases	Percentage of all Diseases
1838–42	12,088	5·78	·69
1843–46	vacat	—	—
1847–51	16,747	6·71	·83
1852–56	17,246	6·71	·83
1857–61	17,250	6·36	·83
1862–66	19,818	6·80	·81
1867–71	23,078	7·51	·93
Total of Thirty Years	106,427	6·48	·82

It is seen from this table that there has been a rise in the mortality from cephalitis ever since registration was commenced, with the only exception of the fourth lustrum, in which there was a slight fall.

The influence of age and sex upon the production of cephalitis is considerable. The annexed diagram shows that the *infantile period of life is particularly subject to such inflammatory conditions*, the first year showing a maximum of 448, and the first lustrum one of 1,414. From that time there is a decided fall, with the only exception of the period from 35 to 40, when a slight rise is noticed; which is, however, again succeeded by a fall until the end.

DIAGRAM F.

Showing the influence of AGE on the Mortality from CEPHALITIS in England and Wales in 1847.



Males are throughout more liable to die of this disease than females, as shown in the following table :—

Periods of Five Years	Males	Percentage	Females	Percentage
1847–51	8,424	3·52	7,541	3·14
1852–56	9,808	3·80	8,128	3·12
1857–61	9,529	3·50	7,721	2.84
1862–66	10,992	3·75	8,816	3·01
1867–71	12,655	4·09	10,423	3·37
Mean Percentage for 25 Years : Males, 3·76 ; Females, 3·10.				

Under the heading of Cephalitis I shall now proceed to consider the following pathological conditions :—

1. *External cerebral pachy-meningitis*, or inflammation of the external layer of the dura mater of the brain.
2. *Internal hæmorrhagic pachy-meningitis*, or hæmatoma duræ matris.
3. *External spinal pachy-meningitis*, or inflammation of the external layer of the dura mater of the spinal cord.
4. *Internal spinal pachy-meningitis.*
5. *Cerebral lepto-meningitis*, or inflammation of the pia mater of the brain.
6. *Spinal lepto-meningitis*, or inflammation of the pia mater of the spinal cord.
7. *Encephalitis*, inflammation of the brain.
8. *Myelitis*, inflammation of the spinal cord.

It will be seen that I have omitted from this list the disease called 'arachnitis,' which occupied a large space in the older text-books of medicine; but it is now generally admitted by pathological histologists that what used to be called the parietal layer of the arachnoid does not exist; and that inflammation of the so-called visceral layer of that membrane never occurs without simultaneous inflammation of the pia mater; so that an independent disease 'arachnitis' can no longer be admitted into our system of nosology. I have also omitted tubercular meningitis or hydrocephalus, which belongs to the class of tubercular affections, and epidemic cerebro-spinal meningitis, as being a zymotic disease.

1. *External cerebral pachy-meningitis*, or inflammation of the external layer of the dura mater of the brain, is generally a surgical disease, and occurs under two forms, viz., the purulent and non-purulent form.

The *purulent form* occurs after injuries by which the skull-bones are fractured and perforated, such as gunshot-wounds, sabre-cuts, etc., and where caries of the petrous portion of the temporal bone, with inflammation of the labyrinth and the cavity of the tympanum, spreads to the dura mater. The tissue of the membrane under such circumstances becomes looser and softened; crowds of white blood-corpuscles emigrate from the bloodvessels, and are found dispersed throughout the tissue of the dura, and they accumulate after a time at certain points, where pus is ultimately discovered. This process is facilitated by transudation of serum from the bloodvessels. There is, however, rarely any very great accumulation of pus, owing to the scanty bloodvessels of the membrane. The inflammation generally spreads to the internal layer of the dura mater, producing internal pachy-meningitis, and also to the pia mater, causing lepto-meningitis.

The symptoms in such cases are not very definite, as they are complicated with those of the injury or disease which gave rise to them, and also with simultaneous affection of the brain itself. Where caries of the internal ear gives rise to this inflammation, there are generally rigors, and the discharge from the external ear is often, though by no means invariably, arrested. There is also headache, vertigo, restlessness, delirium, convulsions, vomiting, constipation, and ultimately coma. Metastatic inflammation of the lungs is likewise of frequent occurrence under such circumstances. In purulent pachy-meningitis after injury, it is of the utmost importance that the pus should be freely evacuated; if this can be done, the inflammation of the dura is often easily borne, and heals by the formation of granulations.

Non-purulent external pachy-meningitis may also occur after injury; but it is observed chiefly in those cases where there is no perforation of the skull, and the membrane has only been separated from the internal surface of the skull-bones by a blow, or similar violence. This separation takes place at the

point where the injury was received, and likewise often at a point opposite to it. Sometimes there is a considerable effusion of blood, which by pressure on the brain may produce serious symptoms. The inflammation is generally not extensive, but remains circumscribed. The tissue of the dura appears reddened by hyperæmia of the bloodvessels, and punctuated extravasations of the blood; crowds of white blood-globules accumulate in the tissue, which appears thickened and swollen, and are first changed into spindle-cells, and afterwards into bundles of connective tissue. In this way the dura ultimately becomes thickened, and adheres firmly to the skull, by the formation of pseudo-membranes. Chalk is not unfrequently deposited into the young connective tissue, which may ultimately become ossified.

External pachy-meningitis is frequently found, without previous injury, in the aged. Here there is a great proliferation of connective tissue; the adventitia of the arteries appears thickened; the membrane is found to adhere to the skull. This chronic form of the disease is often discovered at autopsies, without there having been any particular symptoms indicating it during life. In other cases the patients have for years complained of prolonged headaches; but this kind of headache is also found in atrophy of the brain, with consecutive hydrocephalus, and in chronic inflammation of the pia mater, so that its occurrence does not justify us in making the diagnosis of pachy-meningitis.

External pachy-meningitis is also found in syphilitic disease and erysipelas of the face, but has no special significance in these conditions, as there are always morbid processes of more importance going on in other organs at the same time.

2. *Internal Hæmorrhagic Pachy-meningitis, Hæmatoma Durœ Matris.*

This is pathologically as well as clinically a most interesting disease, which has engrossed the attention of the best observers from the time of Abercrombie and Andral up to our own time. It was formerly believed to be owing to hæmorrhage between the dura mater and what used to be called the 'parietal layer' of the arachnoid membrane; but Virchow was, by numerous

and careful observations, led to the conclusion that the disease is essentially a chronic hæmorrhagic inflammation of the internal layer of the dura, and that all later changes and deposits originate from the false membrane itself. The first thing observed is hyperæmia of the dura, especially of that portion of it which covers the convexity of the brain, and corresponds to the sphere of the middle meningeal artery. The arteries and capillary vessels are distended; a loose yellow deposit consisting of delicate connective tissue is thrown out by the sub-epithelial layer of the membrane, and is seen to contain a number of hæmorrhagic points. This layer is traversed by a large number of bloodvessels which receive their supply of blood from the dura, and can be separated from that membrane. When this is done, large numbers of small bloodvessels which proceed from the dura to the false membrane are ruptured. At the same time serum is thrown out into the sub-arachnoid space, leading to compression and wasting of the brain. The capillaries of the false membrane have three or four times the diameter of ordinary capillary vessels, and by their rupture give rise to repeated attacks of hæmorrhage. From time to time fresh attacks of inflammation take place; the effused blood is again changed into false membranes, from which further hæmorrhages occur. All these membranes are exceedingly vascular; and by the constant repetition of the same process ultimately enormous masses of blood are effused, and the hæmatoma is formed.

This hæmatoma is really a cyst, the external layer of which adheres firmly to the dura, and the cavity of which is filled with a variable quantity of serum and bloodclots. As a rule, such cysts are round and flat, and are found particularly on the anterior and middle portion of the convexity of the brain, while they are much less frequently met with on the posterior part of it. By pressure on the brain the hæmatoma causes the grey matter to become softened and discoloured, and the pia mater and arachnoid are rendered opaque.

Virchow's views were generally received by the profession, but quite recently Huguenin [1] has rendered it probable that the

[1] Ziemssen's 'Handbuch der Pathologie und Therapie.' Leipzig, 1876. Vol. xii. part i. 1, p. 347.

old views of Abercrombie and Andral may after all have been more correct. He denies that there is any real inflammation in the commencement of the disease, and looks upon the process as a truly hæmorrhagic one. According to Huguenin the first stage of this so-called pachy-meningitis is formed by the appearance of a thin layer of dark-red coagulated blood, which has its greatest thickness, viz., about two millimètres, on the parietal tuberosity, and from there spreads equally towards both sides, gradually decreasing in thickness until it quite disappears. Under this layer the dura mater is absolutely unchanged; its vessels are not distended, and the epithelial layer of the inner surface of the dura and of the external surface of the arachnoid membrane is still clearly perceptible. The white blood-corpuscles are arranged in groups, forming patches and trabeculæ, but there are no connective-tissue-cells nor newly-formed bloodvessels. Later on this hæmorrhagic layer begins to look more like a membrane; its colour is changed from red to a brownish yellow; the red corpuscles are still partly intact, but have partly given off their colouring matter, and lost their clear contours; masses of fine brown granular pigment are formed, and the patches of white blood-globules become changed into bundles of spindle-cells, by elongation of their two opposite extremities. The formation of the false membrane is, according to the same observer, a much later phenomenon, and takes place along the falx cerebri and the sinus longitudinalis on the convexity of the brain, from where it spreads to the frontal and occipital lobe and the fossa of Sylvius. In the majority of cases, viz., fifty-six per cent., the hæmatoma was found on the surface of both hemispheres, while in forty-four per cent. the pathological process was confined to the surface of only one hemisphere. In case Huguenin's observations should be confirmed by other observers, and thus shown to be the rule rather than the exception in this condition, it would be necessary to remove the disease under consideration from the inflammatory affections of the nervous centres, and range it under the designation of meningeal hæmorrhage.

What is the source of this peculiar form of hæmorrhage? Huguenin thinks it due not to inflammation but to degeneration and rupture of the veins, which proceed from the surface

of the brain to the longitudinal sinus; since he has frequently found these vessels dilated and varicose, and their coats attenuated and fragile. This would coincide with the wasting of brain which is generally observed at the same time. The vacuum which is made by atrophy is filled up by effusion of serum into the meshes of the pia mater and the ventricles of the brain. The veins are, therefore, greatly distended by blood, even where there is no congestion in other parts of the system.

The effects of the hæmatoma duræ matris on the brain are purely mechanical. Where the false membrane is thin and delicate, it exerts only a very slight pressure on the underlying structures; but where it is extensive, the corresponding portions of the hemisphere appear flattened, the convolutions compressed, and the ventricle small. The falx is pushed over to the opposite side, so that the other hemisphere also becomes compressed. The bloodvessels contain little or no blood, and the brain therefore appears shrunk and wasted; its weight is reduced; and we sometimes find atheroma of the basilar and intra-cerebral arteries, and that form of diffuse chronic encephalitis which leads to general paralysis of the insane.

Hæmatoma of the dura mater is chiefly met with in the inmates of workhouses, lunatic asylums, and hospitals for the incurable. It is never seen in healthy persons, but occurs in the old, decrepit, worn-out, intemperate; and is not unfrequently associated with low forms of pleuro-pneumonia, pneumothorax, pleurisy, pericarditis, emphysema, rheumatic fever, delirium tremens, leukæmia, pernicious anæmia, scurvy, hæmophilia, and the various forms of Bright's disease of the kidneys. It has occasionally become developed in the course of whooping-cough, from excessive venous congestion brought on by severe paroxysms of coughing, and injury to the head may also lead to it.

The influence of the habitual use of alcohol on the production of this disease is admitted by all observers; and Kremiansky has artificially caused this condition in dogs which he fed systematically with alcohol for a considerable time. Kremiansky's experiments, however, seem to lose somewhat in value, inasmuch as Ruge, who repeated them, was unable to produce either hyperæmia of the middle meningeal artery—which Kre-

miansky thought pathognomonic in these cases—or pachy-meningitis. Lancereaux has endeavoured to explain the effects of alcohol in the production of hæmatoma by assuming that it evaporated on the surface of the membranes of the brain, and amongst them of the dura, which it irritated; but Huguenin seems nearer the mark in thinking that the primary effect of habitual alcoholism is wasting of the brain with vascular degeneration, which latter in its turn leads to hæmatoma.

The symptoms of the disease vary considerably, according to its different stages. In the initial period they are generally indistinct, and overshadowed by the simultaneous occurrence of other important diseases, which attract more attention. It is, however, known that the initial symptoms are severe headache, a constricted pupil, restlessness at night, and occasionally convulsions.

The second more chronic period, which embraces the time from the first formation of the hæmatoma until its rupture, may last for years. There is permanent fixed headache, vertigo, impaired memory, indifference to the events of life, tottering gait, stammering, aphonia and aphasia, facial palsy, voracious appetite, and constipation.

The last period, which is characterised by rupture of the hæmatoma, shows the symptoms of apoplexy; but these differ from ordinary apoplexy by their tardy development, so that the patient may be in a semi-apoplectic condition for eight or ten days, sometimes getting a little better, and sometimes worse, as the hæmorrhage from the ruptured cysts increases or diminishes. There is stupor, loss of consciousness, hemiplegia, or general paresis of the limbs. The face is distorted and partially contracted; there is difficulty of deglutition and respiration, and sometimes death is preceded by delirium and convulsions.

Hæmatoma of the dura occurs generally after fifty years of age, and is much more frequent in men than in women.

3. *External Spinal Pachy-meningitis.*

Inflammation of the external layer of the dura mater of the spinal cord occurs almost invariably by a propagation of inflammatory irritation from the neighbourhood of that membrane.

Abscess of the psoas and of the muscles of the back, and decubitus of the sacrum with subsequent caries of the vertebræ, seem to lead most frequently to it. The membrane appears thickened, and its layers interspersed with inflammatory products, which appear either as pus, or connective-tissue-cells. The cord is compressed by these effusions, and appears flattened and anæmic; and this anæmia adds certain symptoms of disease to those which arise from the inflammation of the dura mater. The chief signs of this affection are pain and stiffness in the back and limbs, more especially on moving them, a feeling of tightness round the waist, and hyperæthesia of the skin. If the cord eventually becomes softened from compression and anæmia, paralysis of the extremities with decubitus is the result.

4. *Internal Spinal Pachy-meningitis.*

Inflammation of the internal layer of the dura mater of the spinal cord occurs in two varieties, viz., the *hypertrophic* form, in which there is great proliferation of connective tissue and consequent undue development of this layer of the membrane; and the *hæmorrhagic* variety, which is analogous to the hæmatoma of the dura mater of the brain.

The hypertrophic form of this disease, which has been chiefly studied by Charcot, appears to come on in consequence of living in damp rooms, under bad hygienic influences, and the habitual influence of alcohol. It affects only the cervical portion of the cord, and shows two distinct stages, the first of which is characterised by irritation, and the second by depression of nervous force. These correspond to the anatomical alterations which take place in the membrane. At first there is hyperæmia, and effusion of fibrine which is gradually changed into connective tissue, so that the internal layers of the dura mater are proliferated. The inflammation often extends to the pia mater, which also becomes thickened, and the membranes then adhere to each other, and form an unbroken mass of connective tissue; but in other cases the arachnoid and the pia mater remain unchanged.

The first, or irritative stage, generally lasts from two to three months; and as the cervical portion of the cord is

affected, the symptoms are all referable to that region. The neck is stiff; the upper portion of the chest feels tight; there is more or less constant pain at the back of the head, the nape of the neck, and in the shoulders, arms, and fingers. Sensations of pins and needles and numbness in the upper extremities, together with muscular debility, are likewise complained of; and trophic changes in the skin, such as herpes and bullæ, are not uncommon.

The second, or paralytic stage, is characterised anatomically by the effects of pressure of the hypertrophied membrane upon the nervous matter enclosed by it. The cord, instead of being freely movable in its case, is constricted as if by a vice; its tissue becomes anæmic and softened, or there may be active inflammation extending through the entire width of the organ. The roots of the cervical nerves are likewise constricted and become atrophied; and the muscles animated by them undergo the usual process of granular degeneration.

The symptoms again closely correspond to these anatomical lesions. The upper extremities become paralysed, and more especially is this the case with the muscles animated by the median and ulnar nerves, while those under the influence of the musculo-spiral are less affected. In consequence of this the paralysed limbs are seen to be in a state of extension. The faradic excitability of the muscles is diminished or lost; the galvanic excitability continues much longer, but also disappears eventually as the wasting of the muscular substance progresses. There is more or less complete anæthesia of the skin, so that the highest degrees of faradic power applied by the wire-brush are imperceptible, while the continuous current may still produce a sensation of tingling and pricking to the end, when no other kind of stimulus produces the slightest effect. As time goes on, the pathological process is apt to descend to the lower portion of the cord, causing paralysis and contractions of the lower extremities, with paralysis of the bladder, and decubitus. When this is fully established, the fatal issue is near; yet the progress of the disease is almost invariably protracted; and where it remains confined to the cervical cord, life may be prolonged to its ordinary span.

The second, or hæmorrhagic form of internal spinal pachy-

meningitis, corresponds closely to the hæmatoma of the dura mater of the brain. There is a large quantity of hæmorrhagic effusion, and blood-cysts are formed which contain decomposed blood and pigment. Whether inflammation or hæmorrhage is the primary process, is as yet undecided. The course of this variety of the disease is likewise very tedious. The principal symptoms are pain and stiffness in the back and limbs, muscular debility, and ultimately paraplegia, with paralysis of the bladder and decubitus.

5. *Lepto-meningitis Cerebralis, Inflammation of the Pia Mater of the Brain.*

The non-tubercular inflammation of the pia mater of the brain is either chronic or acute. *The acute form* generally affects the convexity of the hemispheres, and one side more than the other. Where it is partial, the chief seat of the disease is on the convexity of the anterior lobes. The pia mater of the cerebellum mostly remains unaffected, while the inflammation is apt to spread to the corresponding membrane of the spinal cord.

The anatomical features of the acute form of meningitis are as follows:—the finest vessels of the pia mater appear injected and congested. There is an effusion of granular fibrine and emigrated white blood-corpuscles into the sub-arachnoid space, between the convolutions, and in the neighbourhood of the larger bloodvessels. This effusion looks either turbid or yellow, and assumes the characters of pus. The pus is seen to accompany chiefly the large and small veins which course in the parietal regions, and proceed into the longitudinal sinus. The cortex of the brain may remain healthy, but is sometimes subject to inflammatory softening, so that part of the cineritious substance is torn off when the pia mater is removed. The white matter is generally rendered anæmic by the pressure of the effusion, and the ventricles are dry and empty. The effused matters may eventually become completely absorbed, but as a rule the pia remains thickened and opaque.

Where the process has become chronic, the arachnoid is found adherent to the dura, the pia thickened by proliferation

of connective tissue, the subarachnoid spaces filled with opaque liquid, the cortex and ultimately the entire brain wasted; and the ventricles are filled with serum. The pia mater is, under these circumstances, intimately connected with the cineritious substance by means of numerous newly-formed bloodvessels and connective tissue.

Acute lepto-meningitis occurs primarily through the influence of cold and wet, and by exposure to the heat of fire and the sun; but it is more frequently secondary to disease or injury of the skull-bones and the dura mater, such as pachy-meningitis, otorrhœa from caries of the petrous portion of the temporal bone, and inflammation of the sinuses of the dura mater. It also follows inflammation and other diseases of the brain; and where it occurs in advanced life, is often found in decrepit persons, in the habitually intemperate, or comes on during convalescence from pneumonia, pleurisy, pericarditis, typhoid fever, and through the influence of pyæmia, syphilis, and Bright's disease.

The symptoms of meningitis vary considerably, according as it comes on primarily, or in consequence of other diseases. In the latter case there may be nothing but great depression of the vital powers and paralysis; while where it is primary, it is usually ushered in by rigors and heat, with increased temperature and a quick pulse.

The traditional description of the symptoms of meningitis is not quite true to nature. It used to be said that there were two stages—one of irritation, and another of depression; and that the beginning of the inflammation was attended by excitement, while after the effusion had taken place, there was stupor and paralysis. But the several phases of the inflammatory process are not so sharply defined as some authorities would lead us to believe; for we may have different stages of it at the same time in different portions of the diseased membrane, since the inflammation is apt to proceed gradually from one point to another.

In infants meningitis generally commences after a restless night, with a long convulsive fit, and high fever. There is tension and pulsation of the large fontanel. After the fit is over, the children remain somnolent and feeble; a succession of convulsive seizures takes place, and paralysis and coma follow.

Older children complain of headache and photophobia, scream and vomit, and remain excited to the last. Recovery is rare during the infantile period of life. Very young children generally die within thirty-six hours; while those who are older may live on for a week or a fortnight. Where the disease does not kill quickly, chronic meningitis remains, with alternate intermissions and exacerbations; and even then a fatal issue is generally the ultimate result.

In adults the symptoms are sometimes those of maniacal excitement without increased body-heat or other symptoms of a febrile affection. The patients are restless, unable to sleep, and subject to hallucinations and muttering delirium, which is rapidly followed by convulsions, general tremor, paralysis, coma and death. In such cases the cineritious substance of the hemispheres partakes largely in the inflammation.

Where the disease is more confined to the pia mater, headache is one of the most constant and persistent symptoms. It is exceedingly severe, so that the patients continually moan and scream; and even when consciousness is lost they are seen to put their hands to the head with an expression of agony. Sometimes stitches through the head, or a feeling as if the head was screwed down by a vice, are complained of. There is increased reflex excitability, and great hyperæthesia. Tinnitus aurium and sparks before the eyes are common. Slight stroking with the fingers over the skin produces circumscribed erythematous redness. After a time the sensibility becomes dull, and there is general depression. Questions are not understood or slowly answered; there is indifference, stupor, incessant tremor, and not unfrequently convulsions. Stiffness of the neck by contraction of the trapezius is a characteristic symptom. The limbs are relaxed and paralysed, but there is rarely hemiplegia or paralysis of the sphincters. In some cases there is paralysis and contraction of the facial muscles, giving to the face a most repulsive expression. Occasionally the patients do not seem to be very ill when in bed; but if made to get up and walk, they stagger and fall, and all other symptoms are increased.

Signs given by the pupils are puzzling, as is so frequently the case in nervous diseases. They are generally at first constricted and afterwards dilated, but they may be either small or large

throughout the whole course of the disease. Sometimes they are unequal in size, or moderately dilated while entirely unaffected by light. This latter circumstance is often found shortly before dissolution.

The pulse is at first frequent, large, and hard; but after a time becomes very slow, falling from 140 and more to 60 and less. Towards the end it again becomes very frequent, small, and irregular.

Vomiting is nearly always present at the commencement, but now and then appears likewise in the later stages of the disease. There is also thirst and loss of appetite, and the abdomen appears drawn in.

The temperature is at first increased, and afterwards lowered, ranging between 94° and 104°.

Recovery from meningitis is rare; but there can be no doubt that patients occasionally get quite well of it, as traces of previous extensive lepto-meningitis have been found at autopsies of persons who died from totally different diseases. Death sometimes occurs suddenly in the acute as well as in the chronic form of the disease. Favourable signs are the cessation of delirium and restlessness, a quiet sleep, and improved articulation and perception of things on awakening from it.

Chronic meningitis is either the sequela of the acute form of the disease, and is then marked by less violent symptoms and a more tedious course; or it comes on without a previous acute attack, and then generally shows the features of general paralysis of the insane. (P. 164.)

6. *Spinal Lepto-meningitis.*

Inflammation of the pia mater of the spinal cord occurs either by itself, or together with cerebral lepto-meningitis; and may be either acute or chronic.

The acute form also affects more especially children and the male sex, and is frequently caused by cold, such as a fall into the water, more especially in winter; sleeping on the damp ground when the body is heated; living in damp rooms under unfavourable hygienic conditions, &c. Sometimes the inflammation spreads from the dura to the pia, or is owing to caries

of the vertebræ; decubitus of the sacrum which perforates the sacral canal; inflammation in the sac of spina bifida; injuries causing fracture or dislocation of the vertebræ, and simple concussion of the spine. It is also observed during convalescence from acute diseases such as pneumonia, rheumatic, typhoid, and puerperal fever, and cholera.

The anatomical lesions are analogous to those observed in cerebral lepto-meningitis. There is at first hyperæmia; the pia appears congested and swollen, its colour varying from pink to purple; there are capillary hæmorrhages and ecchymoses; the vessels are distended, and the spinal liquid is opaque. Then transudation of serum takes place into the subarachnoid space, and this serum soon becomes purulent. The effusion is more localised where it arises from disease of the vertebræ or other neighbouring structures; otherwise it will spread throughout the length of the cord, of which it occupies chiefly the posterior surface. The roots of the spinal nerves partake in the inflammation; they appear softened and swollen, their fibres are indistinct, and the cylinder axis is granular and thickened. The cord itself is either compressed by the effusion, and then appears pale and anæmic, or it participates in the inflammation in a similar manner as the cineritious substance of the brain takes part in cerebral lepto-meningitis. There is then either interstitial myelitis, with granular infiltration of the neuroglia, which may occur in patches or throughout the organ, or the entire mass is inflamed, so that we find parenchymatous myelitis. In cases which do not prove fatal, the products of inflammation are ultimately absorbed, but some sequelæ generally remain, such as opacity and thickening of the soft membranes, which adhere to each other and the dura; there is hydrorrhachis, i.e. accumulation of liquid in the subarachnoid spinal space, and sclerosis and atrophy of the cord, more especially of its posterior columns.

Not only in the anatomical lesions, but also in the symptoms, the greatest analogy exists between cerebral and spinal lepto-meningitis. There is in many cases a rigor followed by fever-heat, the pulse being full, hard, and quick, beating at the rate of 120 to 140, while the thermometer runs up to 103° and 104°. The urine is scanty, and deposits abundant lithates. There is

intense back-ache, which is chiefly felt on the part from where the inflammation starts, but afterwards extends throughout the length of the spine. Occasionally, however, the commencement is more latent; there is no or only a slight increase of temperature, but a feeling of general malaise and lassitude, which is followed by pain in the back, sensations of pins and needles in the limbs, constipation and difficulty in emptying the bladder.

In acute cases the pain in the back is most severe; it is much increased by movements, such as sitting up or turning round in bed, emptying the bladder and the bowels. It is often not confined to the back, but shoots into the extremities and the front part of the body. Sometimes there is such a degree of rigidity of the muscles of the vertebral column that opisthotonos is produced, the head being drawn backwards and the spine arched; there may also be convulsions in the limbs, similar to those of tetanus, but differing from them by not being brought on through touching the patient or otherwise exciting his sensibility.

The symptoms vary considerably according to the anatomical seat of the inflammation. Where cerebral meningitis precedes the spinal inflammation, the symptoms show a downward course, so that the upper extremities suffer previous to the lower ones; but in other cases the pia mater of the lumbar portion of the cord is first affected, and the inflammation then gradually creeps upwards to the cervical spine and the medulla oblongata.

Cerebral symptoms, such as vomiting, delirium, coma, &c., are only observed where spinal is complicated with cerebral lepto-meningitis; and where the medulla oblongata is affected, there is difficulty of articulation, deglutition, and respiration. *Cervical meningitis* is attended by great rigidity of the neck, pain shooting into the arms and hands, dyspnœa from compression of the origin of the phrenic nerve and rigidity of the diaphragm, difficulty of deglutition, various alterations in the size of the pupils—dilatation, constriction, and inequality—and signs of disturbed innervation of the heart. In *dorsal meningitis* the pain shoots into the body; the chest-walls are rigid, and there is a feeling of constriction round the waist. In cases of lumbar meningitis the patients complain of pain in the loins, sacrum, hypogastrium, and lower extremities, a feeling of great

stiffness at the bottom of the back, and stiffness of the muscles of the lower extremities, which renders them immovable. The abdomen is hard and drawn in, and there is obstinate constipation from spasm of the muscular coat of the bowel, abdominal muscles, and sphincter ani. The bladder is similarly affected, as is shown by dysuria, irritability of the viscus, and retention of the urine.

Reflex excitability is at first increased, afterwards diminished, and ultimately lost. Where the posterior roots are much affected, there is great hyperæsthesia of the skin, muscles, and joints.

In the further progress of the disease, symptoms of paralysis predominate. The limbs are now motionless more from weakness than from pain; there is anæsthesia of the skin, incontinence of urine and fæces, an innumerable pulse, a temperature of 104° and more, and death takes place by asphyxia, from rigidity of the respiratory muscles, and consequent carbonisation of the blood. In hyper-acute cases the patients may die within a few days from the commencement of the disease; but such instances are exceptional. As a rule, death takes place in two or three weeks, either from exhaustion through pain, sleeplessness, and insufficient oxidation of the blood, or from pneumonia and cystitis. In other cases a temporary improvement sets in, which is soon after followed by a fresh exacerbation; or there is a sudden crisis, with great fall of temperature, retardation of the pulse, profuse perspiration, abundant excretion of urine, and epistaxis, bleeding from piles, or menstruation; and the principal symptoms of the complaint then diminish or disappear rapidly. Many cases, however, drag on wearily, getting now better and then again worse; but the patients do not rally thoroughly, and death occurs ultimately, after many months of suffering, from decubitus and blood-poisoning. Finally, there may be recovery as far as life is concerned, but the patients are greatly emaciated, have a tedious convalescence, and remain subject to partial or complete anæsthesia and paraplegia, and where the posterior columns have been principally affected, to progressive locomotor ataxy.

Occasionally the course of the disease is chronic from the very commencement. Such is the case chiefly in persons whose

systemic powers are exhausted by excessive drinking, smoking, and indulgence in the sexual appetite, or who have undergone great fatigues and privations, especially in time of war, where they are also much exposed to the influence of cold and damp. Under these circumstances spinal meningitis is usually complicated with myelitis, leading to sclerosis. There is then no suppuration, but great venous congestion and proliferation of connective tissue; the membranes of the cord adhere to each other and the cord; the latter is frequently found indurated in its entire extent, and the spinal nerve-roots are pale and wasted.

This form of lepto-meningitis commences very gradually with a feeling of heaviness in the lower extremities, difficulty in walking, and pins and needles in the legs and feet. There is tightness round the waist, difficulty of urination and defæcation, and pain and stiffness in the back. The weakness eventually merges into a more or less considerable degree of paralysis. A peculiar feature is that the degree of the palsy varies a good deal from time to time, most probably in consequence of variations in the quantity of cerebro-spinal liquid and the degree of venous congestion that is present. It is in such cases that blistering is of wonderful service. Where there is much venous congestion, the paralysis is worse after the night's rest, and improves by moving about and standing, in the daytime; while where there is an excess of cerebro-spinal liquid, the motor weakness is greater in standing, because the liquid is then not equally divided, but gravitates downwards and compresses the lower portion of the cord more effectually.

The inflammatory process may, in such cases, remain for years confined to the lower portion of the dorsal and the lumbar portion of the cord; and unless recovery ensues under proper treatment, is apt to become aggravated in course of time. The patients often die of marasmus, decubitus, or cystitis; or the disease spreads to the cervical portion of the cord and the medulla oblongata, producing numbness and loss of power in the upper extremities, together with difficulty of deglutition and respiration. Bronchitis and pneumonia are then apt to set in, or the patient ultimately sinks from sheer exhaustion.

All forms of meningitis, excepting the tubercular, caused in 1874 the deaths of 3,013 males and 2,457 females: in all, 5,470.

7. *Encephalitis* (*Inflammation of the Brain*).

'Inflammation of the brain, with red and yellow softening,' caused in 1874 the deaths of 1,426 males and 931 females: in all, 2,357.

There are few diseases the pathology of which has undergone such striking changes in recent times as encephalitis. The best observers are now agreed that many cases, which were formerly believed to be such of inflammation of the brain, were in reality cases of meningitis, or of softening from thrombosis and embolism; and there can be no doubt that *inflammation of the brain is on the whole rare, and never general but always partial.*

Encephalitis is in the large majority of cases owing to injury to the head, and therefore found chiefly in adult males, who are more liable to accidents than others. In children it may arise from otitis and caries of the petrous portion of the temporal bone, under the influence of the strumous diathesis, or after scarlet fever. Where it occurs after injury, the skull and meninges are generally likewise affected; but where there is simple concussion and no fracture, capillary apoplexy may be the primary result, and this may eventually lead to acute or chronic encephalitis. Erysipelas from injury to remote parts may creep up to the face and head, and give rise to inflammation of the brain. In the same way diseases of the skull—such as syphilis, ozæna, caries—may propagate the morbid process to the cerebral tissue. In the aged, fatty or calcareous degeneration of the arterial coats, and compression of the brain by tumours and aneurisms, will sometimes give rise to it. Finally, certain general diseases, such as pyæmia, septicæmia, the puerperal process, typhoid fever, and glanders, may now and then be complicated with encephalitis. There seems to be a predisposition to this disease in persons who are given to drinking, or subject to disease of the heart, or have undergone much mental anxiety and excessive physical exertions.

The chief seat of the inflammation is the cineritious substance of the cortex and the central ganglia, viz. the corpus striatum and thalamus opticus. In the cineritious substance

the inflammation may be diffuse, but in the other portions of the brain there is generally only one small area, which rarely exceeds the size of a filbert or walnut.

The anatomical lesions are as follows:—there is hyperæmia and capillary hæmorrhage, whereby the cerebral matter assumes a red coloration and becomes softer and moister than usual; the convolutions are swollen and pressed against each other; while the uninflamed cerebral matter in the neighbourhood, and the pia mater, are dry and anæmic. The microscopic examination of the affected parts shows crowds of white blood-corpuscles, nerve-fibres in a state of more or less advanced destruction, decayed red blood-globules, dilated arterioles with thickened and fatty coats, crowds of nuclei, amyloid corpuscles, and pus.

If the patients survive long enough, further changes will be observed in the diseased parts; and these vary according to the quantity of blood which has been effused. Where this has been large, things progress much in the same manner as after cerebral hæmorrhage from rupture of miliary aneurisms (p. 94). The colouring matter of the blood-corpuscles is dissolved, and a dirty-yellow substance is left, which is incapsulated by the gradual formation of a cyst. This is mostly seen in the central ganglia, while in the cineritious matter yellow stripes or callosities are discovered, which consist of the remains of blood-globules and sclerosed connective tissue, and are intimately connected with the pia mater, which appears thickened and opaque. Where, on the other hand, only little blood was effused, we meet in the later stages with a kind of paste or jelly, which contains the remains of the decayed cerebral matter.

Abscess of the brain may also be the result of inflammation. In such cases there is rapid emigration of white blood-corpuscles, and all tissues are changed into a kind of emulsion of variable thickness, which is reddish where much blood has been effused, but otherwise shows a light yellow or greenish tint. Such abscesses occur chiefly in the medullary matter, and vary in size from that of a pea to that of an apple; and there may be only one or a dozen of them. In pyæmia they are apt to be multiple.

Where the morbid process is rapid, the abscess is diffused

and irregularly shaped; the neighbouring cerebral matter is not well separated from it, but there is red and grey softening, and punctuated apoplexy in the neighbourhood, with tendency to spread. The pus ultimately reaches the surface of the brain, or breaks through into the lateral ventricle, whereby the whole hemisphere is transformed into an abscess. Such cases are mostly owing to injury or caries of bones; and as the air has therefore access to the pus, this is apt to become discoloured and offensive.

In other cases the pathological phenomena are more chronic in character, and the abscess then generally assumes a regular oval shape. It is contained in a kind of capsule, which consists of connective tissue and bloodvessels, and is intimately connected with the surrounding cerebral matter. The inner surface of the capsule is quite smooth, and the pus has a variable appearance, according to its age and mode of formation. Such an abscess acts like a foreign body on the brain, producing pressure; the convolutions appear flattened, and the pia mater is anæmic and dry. Rupture of the abscess is always fatal.

There is a peculiar form of encephalitis which occurs in children of parents who have suffered from syphilis or small-pox. In these there is diffuse inflammation of the white substance of the hemisphere, while the grey matter escapes. The cells of the neuroglia undergo fatty degeneration, and granular corpuscles and oil-globules are the ultimate morbid product. This variety of encephalitis occurs either previous to or shortly after the birth of the infant.

There are few diseases in which the symptoms vary so much in character and intensity as they do in encephalitis. Sometimes they are so indistinct throughout the course of the malady that it is impossible to diagnose it. In other cases the commencement only is indefinite and slow, but the cerebral symptoms gradually become more distinct as the disease advances. Again, there may be apoplexy in the beginning, after which the course of the complaint may be protracted; while, in exceptional cases, we have severe inflammatory symptoms from the first, leading rapidly to a fatal result.

Encephalitis has been generally believed to be attended

with fever, but the body-heat usually remains at its usual standard, or rises only very little above it towards evening. The pulse, instead of being accelerated, is generally retarded, and may fall as low as forty beats in the minute; the tongue is coated, the appetite deficient, the bowels confined. Where the cerebellum is implicated, there is generally vomiting, more particularly at the commencement of the affection.

The mental faculties suffer in most cases from the beginning, there being either excitement or depression. Restlessness is frequent; the patients cannot sleep, are constantly in and out of bed, take their clothes off and put them on again, and are always busy about something. Delirium is not infrequent, but maniacal excitement is rare. Sometimes great indifference, forgetfulness, and confusion is shown, and this state of dreaminess gradually merges into somnolence and coma.

The nerves of special sense are generally affected. A common symptom is hyperæsthesia of the retina, causing sensations of stars, sparks, and coloured light; and undue excitability of the auditory nerve, causing tinnitus aurium. Common sensation suffers likewise, there being pains in the arms and legs, or numbness, and sensations of pins and needles. Headache, which is such a prominent symptom in meningitis, is less marked in encephalitis; and if at all intense, denotes that the meninges are simultaneously affected, as is always the case in inflammation after injury or from otitis.

The power of motion is generally impaired. There may be hemiplegia or paralysis of one limb, but in most cases there is rather weakness than paralysis. Tremor is frequent. There may be paralysis of the ocular muscles, ptosis and strabismus, or facial palsy, which mostly arises from compression of the portio dura in the Fallopian canal after otitis. Articulation is imperfect; the patients stammer, shuffle the tongue about in a peculiar manner, and rarely finish a sentence. They are awkward in using their hands, and the gait is tottering. Partial convulsions of the limbs are often observed, and towards the end regular epileptiform seizures take place.

Where encephalitis is owing to injury, the symptoms are generally mixed with those of meningitis, more especially where the morbid phenomena become rapidly developed. In some of

these cases the patients may appear perfectly well for eight or ten days after the accident, when suddenly formidable symptoms of cerebral disturbance come on, which either lead rapidly to a fatal issue, or may last for many months, with alternate intermissions and exacerbations.

Inflammation of the brain from otitis is preceded by headache, discharge of sanious pus from the external meatus, fistulous openings in the neighbourhood of the ear, and deafness. Thrombosis of the sinuses of the dura mater and pachy- and lepto-meningitis are commonly associated with it. In these cases the inflammation in the brain is mostly limited to the posterior lobe and the cerebellum of the same side; while where ozæna gives rise to it, the frontal lobe of the brain is more apt to suffer. The onset may be gradual or sudden, according to the intensity of the intracranial lesions. There is fever, vomiting, headache, convulsions, somnolence, and coma. The pulse is either very frequent or very slow, and death may occur very suddenly.

Where pyæmia leads to encephalitis, the progress of the disease is very rapid. This is seen after phlebitis, in the puerperal state, and after otitis. There are rigors, followed by intense fever-heat, with delirium, which quickly subsides into coma and death.

In idiopathic encephalitis of the aged, the symptoms may be protracted over months and years. The patients suffer from tremor, incomplete paralysis with contraction, epileptiform seizures, and impairment of the mental faculties. They show great disinclination to work; the temper is irritable and depressed, the thoughts confused, and the speech drawling. The face has a peculiar expression of astonishment. Headache, giddiness, restlessness at night, and difficulty in walking, are complained of. Ultimately the patients become bedridden; epileptic and paralytic fits succeed each other, and death is, as usual, preceded by delirium and coma.

In *cerebral abscess*, the chief symptom is localised headache, in accordance with the seat of the lesion; and shivering fits and convulsive seizures are not uncommon. Spontaneous abscess is never cured; but where it is owing to injury, the pus may be emptied spontaneously through the opening of the skull, or

burst through the nose or ear, or be evacuated by trepanation; after which there may be complete recovery.

Encephalitis is almost always fatal, more especially where it is idiopathic, or where the inflammation spreads from neighbouring parts. Only where it is owing to injury, there are prospects of recovery. A quantity of brain-matter may be lost without much harm, provided there be complete cicatrisation and separation from healthy parts; for certain portions of the brain are not absolutely essential to life, and their function may be transferred to neighbouring parts. A physiological illustration of this fact is afforded by the experiments of Nothnagel and Goltz, who found that, after experimental destruction of certain portions of the brain, there was loss or impairment of certain motor functions; but that within a few weeks, more or less complete recovery of the function had taken place (p. 49).

8. *Myelitis.*

This disease caused, in 1874, the deaths of 160 males and 133 females. 'Spinal marrow disease' caused, in the same year, the deaths of 64 males and 61 females.

Inflammation of the tissue of the spinal cord is much more rare than meningitis, and occurs either complicated with the latter disease (meningo-myelitis) or by itself. Its causes are the same which produce meningitis; and amongst these the influence of wet and cold stands uppermost. An interesting physiological illustration of the influence of intense cold on the spinal cord is furnished by Feinberg's experiments, in which the prolonged application of ether spray to the spine of rabbits caused paraplegia, which was in some cases temporary, while in others death ensued, which was found to be owing to inflammatory softening of the posterior columns of the cord.

Clinically cases of acute myelitis are met with in persons who have fallen into the water, more especially in winter, from breaking through the ice; after standing in water for a long time; or after getting wet and sleeping in wet clothes. The next influence is over-exertion, such as forced marches of an army in the field, or lifting heavy loads. Veterinary surgeons are familiar with a form of myelitis in horses, after severe and

unduly prolonged running. Where cold and over-exertion are combined, as is so frequently the case in campaigns, the pathological influence becomes intensified. Myelitis also comes on after violent anger, terror, and other mental emotions; in the course of ague, typhoid fever, pleuro-pneumonia, and puerperal fever; from hæmorrhage into, and tumours of, the cord; from disease of the vertebræ, such as caries and cancer; after injury, such as concussion of the spine and cord, fracture and dislocation of the vertebræ, sabre-cuts and gunshot wounds; and from meningitis spreading to the medullary matter.

The following are the anatomical lesions of myelitis. There is softening of various degrees and extent, which may be distinguished by the naked eye. In slight cases it is barely perceptible, but sometimes it is excessive, so that the marrow appears to be changed into a thin paste. The colour of the inflamed parts varies according to the stage of inflammation which comes under observation. The first stage is that of *red softening*, with hyperæmia, effusion of blood, and œdematous swelling of the affected parts. The second stage is that of *yellow softening* or fatty degeneration of the nervous matter; and the third that of *grey softening*, or absorption and wasting. Purulent softening, or abscess of the cord, would seem to be exceedingly rare in men, but has been experimentally produced by Leyden,[1] with injection of liquor arsenicalis into the cord of cats and dogs.

The microscopic examination of the inflamed parts, after they have been hardened in spirits of wine and a solution of chromate of ammonia, and coloured with carmine, reveals the following pathological appearances:—In the first or *hæmorrhagic stage*, blood is either effused in considerable quantities, or there is hæmorrhagic infiltration into the meshes of the parenchyma. Later on heaps of brownish and yellow pigment are found in the tissue. The veins appear much dilated, the arterioles are rarely distended, but their adventitia shows hæmorrhagic infiltration (peri-vascular hæmorrhage). The nervous matter itself is considerably altered; the nerve-fibres are enlarged, the cylinder axis swollen, aud vascular spaces or vacuoles are encountered. The ganglion cells, particularly

[1] 'Klinik der Rückenmarkkrankheiten.' Berlin, 1875, vol. ii. part ii. p. 125.

those of the anterior grey horns, are also enlarged and swollen: their nuclei are thickened, and their contours indistinct. The structural elements of the neuroglia are at first enlarged and swollen, they afterwards appear to become divided, and a few oil-globules are seen between the fibres.

In the second stage of *yellow softening* or fatty degeneration, there is less hyperæmia; the effused blood has undergone the usual changes; the swelling is diminished; the nerve-fibres become dissociated by oil-globules, appear compressed, reduced in size, and devoid of marrow. This latter and the cylinder axis undergo fatty degeneration; the ganglion cells waste away, the cohesion of the stroma is loosened; portions of the connective tissue become thickened and hypertrophied, while other parts of it disappear; and at last a loose puriform paste is formed, which consists of oil-globules, débris of nervous matter, and connective tissue.

In the third stage of *grey softening*, or absorption, the oil-globules become less numerous, the nervous débris absorbed, and there are large lacunæ containing a clear or turbid serum, small distended veins, and tough connective tissue. In such cases the continuity of the cord may be entirely destroyed, there being an upper and a lower cone left, with no intermediate nervous matter at all. Suppuration and abscess being exceptional, the ultimate result of myelitis is generally sclerosis; but in mild cases the inflammatory products may be absorbed, and a certain amount of regeneration of the nervous matter takes place.

All the different portions of the cord may become the seat of inflammation; but the grey substance is more especially liable to it. It is generally first affected, and the morbid process after a time may spread from the grey to the white matter and to the periphery. Where myelitis arises from disease or injury of the vertebræ and the meninges, it is chiefly peripheral; there are areas corresponding to two or more vertebræ, and the inflammation is found most severe in the centre of such areas, while it gradually vanishes towards their extremities. The most extensive areas are habitually found in the middle and lower portion of the dorsal cord, and the inflammation is here seen to have spread either more in a longi-

tudinal or more in a transverse direction. In the cervical cord and the medulla oblongata the inflammation is rather more discrete, the areas being only of the size of a pea or filbert; and we then speak of insular, multiple or disseminated softening. Under certain circumstances the morbid process, after having been for some time confined to a small area, shows the tendency to travel upwards or downwards, leading to myelitis ascendens or descendens. Ascending myelitis may spread to the brain; and descending myelitis to the nerve-roots, nerves, and muscles, leading to neuritis descendens and myositis. Those parts of the cord which were at first unaffected may eventually undergo Türck's secondary degeneration, without inflammation; and this may likewise spread to the peripheral nerves and muscles, causing various degrees of wasting as their ultimate result.

Myelitis from pressure of dislocated vertebræ or fragments of vertebræ is most frequently seated in the lumbar portion of the cord, and is one of the most dangerous forms of the disease, especially where the injury has been extensive. Complete destruction of the lumbar cord by fracture of vertebræ is almost invariably fatal, while injury higher up is not nearly so dangerous. In cases of lumbar myelitis from pressure, the symptoms are particularly marked in the sphere of the sciatic, and much less so in that of the crural nerves. There is generally complete paralysis of the hamstring muscles, and the legs and feet, while the ileo-psoas and the rectus of the thigh retain their mobility. The anæsthesia is proportionate to the paralysis, being considerable in the ham, about the nates, anus, and urethra, but much less in front of the thigh. The sphincters of the rectum and the bladder are paralysed. The reflex excitability of the muscles of the lower extremities is at first increased, and afterwards lost. The faradic and galvanic excitability of these muscles, and their nutrition, are likewise much diminished; they become flabby and wasted in a very short time. General disturbances of the nutrition of the affected parts also make their appearance, the most important of these being decubitus, which is often accompanied by œdema of the feet and legs.

Spontaneous myelitis may affect all the different portions

of the organ, but is most common in the lower portion of the dorsal cord. There is rarely any fever, i.e. increased body-heat, even where the course of the disease is rapid. In many cases general symptoms are quite absent; the patients look in their usual health, have a good appetite, and sleep well. The pulse is not raised above the normal standard. There is rarely any pain, except where there is complication with meningitis. In exceptional cases, however, the temperature runs up to 101° and 102°, and there are rigors, a frequent pulse, vomiting, abundant perspiration, restlessness, and delirium at night. Sometimes the paralysed limbs have a higher temperature than the rest of the body.

The progress of the disease may be extremely acute, so that the patient becomes paralysed in a few hours or days. In such cases the diagnosis of spinal hæmorrhage is apt to be made, but inflammatory softening may occur almost as rapidly as hæmorrhage. The disease occasionally progresses as it were by fits and starts, there being regular attacks of it which are separated by a few days' interval; and each fresh attack leaves the patient more helpless than he was before. Or the myelitis may follow a subacute course, so that the paralysis, from being at first insignificant, gradually increases in severity, and becomes complete in a few weeks.

The first symptoms are often stiffness and heaviness of the limbs; a feeling of 'pins and needles' as if the legs were going asleep; and a sensation of pressure in the region of the bladder and rectum. In some cases there is great restlessness in the limbs, which are constantly thrown about and cannot be kept quiet. When the affection is seated at the periphery of the cord, an acute dragging pain is experienced, more particularly at night. The 'pins and needles' generally begin in the feet, or, if the seat of the disease is in the higher portions of the cord, in the fingers, and spread from there upwards until they seem to have invaded the whole of the limbs. In meningo-myelitis there is great hyperæsthesia of the skin, so that the slightest touch or pressure is intolerable. Pain, which is confined to the seat of the lesion, and increased by pressure on the vertebræ, may also be present. Finally there is a feeling of tightness and constriction, which, according to the seat of

the disease, is felt more round the waist, or more round the neck.

The principal symptom of myelitis, however, is always paralysis of motion, which corresponds exactly to the seat and extent of the disease. It is first noticed in the feet or hands, and is incomplete, so that the muscles do not properly obey the commands of volition, and the movements are awkward and clumsy. Paresis, however, gradually increases to paralysis, which is accompanied with rigidity, where there is complication with meningitis. Sometimes the toes and feet may be moved, or the knees flexed, when the patient is in bed, yet walking is impossible; or the patient may be able to walk, but the gait is tottering, the feet can hardly be lifted from the ground, support is wanted, and fatigue induced by the slightest exertion.

Inflammation of the lumbar cord leads to paraplegia; where the dorsal cord suffers there is also paralysis of the sphincters, and if the disease is somewhat high up, irregularity of the heart's action. In cervical myelitis the upper extremities alone may be paralysed—a condition which is called *brachial diplegia* or *cervical paraplegia.* Where, however, the entire transverse section of the cord is inflamed, there is complete paralysis and anæsthesia of all parts below the lesion. Myelitis above the origin of the phrenic nerve leads to dyspnœa and cyanosis from paralysis of the diaphragm. If the disease is below the origin of the phrenic nerve, inspiration may still be properly performed, yet there is paralysis of the expiratory muscles, and the patient, who is still able to gape, cannot cough or sneeze. Where the inflammation spreads to the medulla oblongata, or even higher up, respiration, deglutition, and the heart's action are involved, and death is preceded by delirium and coma.

Myelitis is mostly bilateral; but in some cases only one half of the cord may be affected, and then there is *hemiparaplegia* or *spinal hemiplegia.* In accordance with physiological data (p. 24), it is found that in such instances there is paralysis corresponding to the side of the lesion, and anæsthesia on the opposite side. There is, however, a general tendency inherent to this disease to spread to the entire organ, where it commences with cervical paraplegia. Again, the

unilateral variety is apt to progress into the bilateral form, but ordinary paraplegia of the lower extremities has less tendency to spread upwards.

Spasmodic symptoms are also commonly observed. The muscles of the legs particularly are subject to convulsions, combined with shooting pains through the limbs. Fibrillary twitches in the muscles are a later symptom, which shows wasting of the contractile fibre. Sometimes the convulsions of the lower extremities are exceedingly severe (Brown-Séquard's 'Spinal Epilepsy'), and occur spontaneously, or are excited by some kind of irritation, such as the application of cold, tickling of the soles, the introduction of a catheter, or a tap on the muscles. This shows that reflex excitability is largely increased. The limbs may be so violently flexed that the heel touches the hip, or there is complete rigidity of the limbs or of some parts of them. In such cases the seat of the disease is always above the lumbar enlargement.

As a rule, however, the paralysed muscles are completely relaxed, and do not offer any resistance to passive movements. In the later stages of the disease contractions are frequent, more particularly of the adductors and the hamstring muscles. Sometimes there is permanent extension of the knee, resembling Charcot's permanent hysterical contraction.

The state of sensibility varies considerably in the different forms of myelitis. In some cases there are in the commencement of the disease hardly any abnormal sensations, such as pain, pins and needles, or numbness; while in others such symptoms are very striking. This may be explained by the inflammation attacking sometimes more the grey substance in the centre of the cord, and at other times more the periphery of the organ, implicating the pia mater and the posterior roots. *Where there is a moderate degree of inflammation in the grey centre or the antero-lateral columns, no pain is experienced.* In such cases the spine is not unduly sensitive to pressure, percussion, and the applications of heat and the various forms of electricity, nor do movements of the body cause pain. There is, however, frequently a feeling of tightness round the waist, more especially in lumbar myelitis from pressure, and round the neck in cervical myelitis.

Where the posterior roots and the pia are involved, there is pain in the back and the extremities, especially at night. This pain is of a throbbing or shooting character, and often combined with painful tingling and convulsion. Such symptoms, however, disappear in the later stages of the disease, when a feeling of coldness and numbness is the rule.

There is rarely complete anæsthesia, even where there is total paralysis of motion. Hyperæsthesia, increased reflex excitability, and a feeling of numbness are more frequent. Where reflex excitability is lost in a few days or weeks, the affection is severe, and involves the grey substance in the centre of the cord.

The faradic and galvanic exploration of the muscles affords important indications as to their condition and to the seat of the disease. At first the electric excitability is normal, but later on it becomes diminished. This is more particularly the case where the lumbar enlargement suffers, and less so where the seat of the disease is higher up. In severe cases it is often rapidly lost; and we then sometimes notice the curious phenomenon that while faradic excitability is gone, the continuous current may produce abnormally exalted effects. Where faradic or galvanic excitability return after having been absent or considerably diminished, a healing process in the cord may be safely predicted, and recovery of voluntary motion is then generally not slow in following. This condition is therefore important in a prognostic point of view.

The nutrition and electric excitability of the muscles depend upon the state of integrity of the grey centre of the cord, more particularly in the lumbar and cervical enlargements. Where only the white matter suffers, both remain normal; while, when the grey substance is diseased, both suffer, even if the white matter should have remained quite healthy. The condition of the trophic centre in the grey substance of the cord may therefore be recognised with certainty by the state of the nutrition and electric excitability of the muscles. Where these are much impaired, there is great destruction in the grey centre; and where they are normal or nearly so, the centre is shown to have escaped the lesion more or less.

If myelitis be followed by descending neuritis and myositis,

great wasting of the muscles takes place, mostly with total loss of their electric excitability. Sometimes these changes are confined to certain sets of muscles, leading to contraction of the antagonists and more or less considerable deformity of the limbs. The contracted muscles may become hypertrophied, and this is more particularly seen in the ham.

Where the paralysis is not complete, the degree of it is generally greater after rest and less after moderate exercise. It is also more marked where the inflammation affects chiefly the antero-lateral columns, while there is more deficiency in co-ordination and muscular sensibility where the posterior columns are the seat of disease.

Paralysis of the sphincters is one of the most important consequences of myelitis. It is generally preceded by spasm and hyperæsthesia of the bladder and rectum, there being frequent but ineffectual desire to pass water, and complete or partial retention of the urine; and spasm and pain in the rectum, with retention of fæcal matters. In the majority of cases the affection of the bladder is more marked than that of the rectum. After the period of spasm and hyperæsthesia has lasted for a short time, the sphincters become paralysed, with the result that there is incontinence of the urine and fæces.

The paralysis of the sphincters is intimately connected with lesions of sensibility. We have seen in the first chapter (p. 29) that in the normal state, when the bladder is full, a desire is experienced to empty it, and a contraction of the detrusor urinæ takes place by reflex action; but where there is anæsthesia of the bladder, the desire to evacuate the viscus is lost. Urine therefore, unless drawn off by the catheter, accumulates in the bladder until at last the tension of the sphincter is overcome, and the urine begins to dribble away. When this condition is caused, the bladder cannot be completely emptied except by the catheter; and cystitis, or catarrh of the bladder, is the result.

This is produced in the following manner:—When urine accumulates and stagnates in the bladder, an alkaline decomposition of the secretion takes place; triple phosphates are formed, and vibriones and bacteria are found to swarm in the fluid, which also contains muco-pus in more or less considerable

quantities. The smell of the urine is offensive when it is passed, and much worse after standing for some hours. In consequence of the inflamed condition of the mucous membrane, the muscular coat of the bladder loses its tone, and paralysis of the detrusor urinæ is combined with anæsthesia of the viscus. Unless very great care and cleanliness are observed, this condition of the bladder must lead to decubitus; but a still more dangerous consequence of it is, that the inflammation is apt to spread through the ureters into the pelvis of the kidneys and produce pyelitis and nephritis. This again leads to blood-poisoning, which is evidenced by rigors, heat, and hectic fever; and the patient is then carried off by uræmia, pyæmia, or exhaustion.

It was formerly believed that after injury to, and subsequent inflammation of, the cord, the urine was secreted in an alkaline state; but such is not the case. The urine is found to be acid in the pelvis of the kidneys, and only becomes alkaline in the bladder, from catarrh of the mucous membrane of that viscus.

The sphincter ani is at first spasmodically affected, as shown by hyperæsthesia, spasm, and tenesmus. The fæces are ejected suddenly by spasmodic contraction of the muscular fibres; or there is obstinate constipation, with tympanitic distension of the abdomen. Later on the sphincter becomes paralysed, so that involuntary alvine discharges occur, whereby decubitus is powerfully promoted.

Decubitus, *bedsores*, or *local gangrene* occurs in two different forms in this disease. There is first an acute variety, which corresponds in most particulars to the acute decubitus which comes on after cerebral hæmorrhage (p. 104). This is not owing to pressure, or to the contact of the excreta with the skin, but to paralysis of the trophic centre in the grey substance of the cord, and generally forebodes a fatal result. It is combined with an increase of temperature, which is, however, not so great as when the cerebral macula makes its appearance, the mercury rising rarely beyond 101°. The succession of phenomena is otherwise the same as during cerebral fever—that is to say, there is at first a macula, which is soon changed into a vesicle or bulla, resembling herpes or pemphigus. The bulla bursts after a short time, and an ulcer is left which has the

tendency to spread, and occasionally lays open the whole sacrum in a few days. Skin, muscles, ligaments, and bones are all destroyed in a very short time; and this often leads to pyæmia and pulmonary embolism. Where the spinal canal is laid open, acute ascending spinal meningitis may be the result. Thus decubitus may kill in a variety of ways. The acute variety of it is chiefly observed in myelitis from injury, and destruction of the lumbar enlargement. Although most of such cases end fatally, this result is not quite so uniform as it is in the cerebral form of decubitus. Occasionally, indeed, an eliminative inflammation is established, and the sore ultimately heals well. A physiological illustration of this is afforded by the experiments of Goltz and Leyden, who found great tendency to acute decubitus shortly after artificial production of myelitis; but where the animals survived, it soon became considerably diminished. Acute decubitus is observed not only in the sacrum, but also on the hips, knees, and ankles.

An entirely different form of bedsore is that which is developed after myelitis has lasted for a considerable time, and entered the chronic stage. Then local gangrene is slowly developed in those parts which are chiefly subject to pressure. There are two circumstances which promote a long-continued pressure of the body on the bed, viz., the helplessness of the patient, who has the greatest difficulty in changing his position, and is sometimes entirely unable to do so without the aid of an attendant; and secondly, the anæsthesia which is combined with the paralysis, and which enables the sufferer to support the same position without discomfort for a much greater length of time than he could in health. Circulation therefore becomes sluggish, and when irritation by the contact with the excreta is superadded to this, the bedsore is sure to appear. This form of decubitus may generally be prevented by frequently changing the patient's position, scrupulous cleanliness, and the use of stimulating lotions to the parts; while acute gangrene cannot be prevented by any treatment whatever.

Chronic decubitus is characterised by the slow appearance of dry leathery eschars, which are separated after a time by inflammation in the subjacent and neighbouring structures,

and then leave an open sore of varying extent, but mostly very large, with putrefaction and destruction of the affected tissues. It is generally absent in the milder forms of the disease, where there is only incomplete paralysis and anæsthesia, so that the patient is enabled to change his position frequently without assistance, and has enough sensibility left to be disagreeably impressed by the effects of prolonged pressure. In such cases the bladder and bowels likewise often retain their tone, so that a further cause, viz., contact with irritating excreta, is absent.

Other trophic disturbances which occur as a consequence of myelitis, are observed in the skin and the muscles of the affected parts. The wasting of the muscles is, like decubitus, of a twofold character, viz., either more or less acute, from the inflammatory process cutting off the influence of the trophic centre in the grey substance of the cord; or it is more chronic, and then chiefly owing to disuse of the muscles. As long as the muscles are connected with the trophic centre, there is only little wasting, just as in hemiplegia from cerebral hæmorrhage and softening; and atrophy, if there be any, affects chiefly the calves of the legs. Where, however, the centre of nutrition is considerably damaged or destroyed, the wasting is uniform and rapid; and such cases resemble progressive muscular atrophy in its later stages.

This wasting of the muscles may be simple atrophy, and then there is an extraordinary diminution in the bulk of the limbs; or there is an enormous development of fat between the muscular fibres, so that pseudo-hypertrophy of the muscles is produced. The muscular fibres ultimately vanish completely, and the hypertrophied masses are found to consist of fat, connective tissue, nerves and bloodvessels.

The nutrition of the skin is generally much impaired where there is wasting of the muscles. The circulation in the corium and subcutaneous cellular tissue is slow and stagnant; the epidermis appears wrinkled, and is apt to desquamate. The skin is discoloured, showing a dirty yellow brownish tint. There may be an excessive growth of hair, and of the nails, which have to be cut several times a week. The appearance of the nails may also be changed; instead of pink, they look yellow, are clubbed, and break easily. Herpes zoster is not

unfrequently present. The temperature of the limbs which was at first increased, is in the later stages of the disease lowered by 2° or 3°; there is œdema from paralysis of the bloodvessels, and a difference in the perspiration of the paralysed parts and of the rest of the body. Sometimes the body perspires freely, while the lower extremities remain dry and cold; and in other cases there is profuse perspiration in the paralysed limbs, while the rest of the body is quite dry.

Cerebral symptoms, which are common in some other affections of the spinal cord, such as insular sclerosis, and progressive locomotor ataxy in its last stage, are generally absent in myelitis. They may, however, become developed when blood-poisoning takes place in consequence of cystitis or decubitus. Uræmia, or ammonæmia and septicæmia, are generally accompanied by headache, delirium, and somnolence. Where ascending myelitis or meringo-myelitis creeps up to the brain, there are likewise cerebral symptoms, which are at first those of excitement, and afterwards of depression. The patient appears then in a typhoid condition, with muttering delirium and somnolence, which soon deepens into coma. Cerebral symptoms also occur in neurolytic bronchitis, when the phrenic nerve becomes paralysed through caries, cancer, or fracture, and subsequent inflammation of the cervical cord above the third vertebra; and death is then likewise preceded by delirium and coma. The speech is affected where cervical myelitis spreads to the medulla oblongata, and destroys the motor nuclei of the hypoglossus and other cerebral nerves.

The pupils may be contracted, dilated, or unequal in size, not only in cervical, but also in dorsal myelitis. This is explained by affection of the *cilio-spinal region of the cord*, which is situated between the seventh cervical and the sixth dorsal vertebræ. Pathological irritation of this region has the same influence as experimental Faradisation, viz. to dilate the pupil; and this is seen in the commencement of dorsal myelitis. On the other hand, the pupil becomes constricted by destruction of the region, which occurs in the later stages of dorsal myelitis. Inequality of the pupils may be caused when only one lateral half of the cord is diseased, while the other half remains healthy.

Respiration and circulation remain normal in myelitis, unless the pathological process invades the upper portion of the cervical cord, where the pneumogastric and phrenic nerves are exposed to the pathological influence. More especially after fracture of the cervical spine there is not unfrequently neurolytic bronchitis, which sets in on the second or third day after the accident. Respiration is insufficient from paralysis of the diaphragm; it becomes very frequent; mucus accumulates in the air-passages and cannot be expectorated, as coughing is impossible, owing to paralysis of the expiratory muscles. Hyperæmia and œdema of the lungs are then developed, and the patient dies in a state of cyanosis.

The appetite is generally good, and digestion satisfactory. Vomiting appears only to occur from uræmic and other forms of blood-poisoning, after cystitis and decubitus. The lower portion of the bowel is, however, often in a state of catarrh, which causes diarrhœa, accompanied with tenesmus.

Priapism is not a constant symptom of myelitis, but occurs now and then, more especially where the cervical and dorsal cord are affected. It may occur at the commencement or in the further progress of the disease, and last sometimes for several days consecutively, with excitement of sexual desire. It causes much pain and inconvenience, although the erection is as a rule not so complete as it is in health. In the later stages of myelitis, the sexual power is completely lost, and erection impossible.

When myelitis occurs in children, the symptoms of irritation are more marked than in adults. There is much fever, choreic restlessness, clonic convulsions, and sometimes tetanic rigidity of the whole frame. The neck is stiff, the jaw locked, and the issue generally fatal. In the few patients who survive, motor paralysis remains after the attack is over.

Myelitis is always a severe disease, which in a large number of cases proves fatal in two or three days, or as many weeks. It is more dangerous to life when seated high up in the spinal canal. Fracture of the odontoid process of the atlas, and other kinds of injury near the medulla oblongata, cause death almost directly, and before inflammation can be developed; but even where the morbid process is lower down in the cervical spine,

the patient's life is in great jeopardy. Inflammation of the middle portion of the dorsal cord is also dangerous, the spinal canal being very narrow in that locality, owing to which the cord is more easily disorganised in its entirety. In cervical myelitis, death generally occurs from respiratory paralysis, while in dorsal and lumbar myelitis it is more frequently owing to blood-poisoning from decubitus and cystitis.

When myelitis does not kill within two or three weeks, its progress becomes of a more chronic character. The patient is even then by no means out of danger, more especially where the affection has come on spontaneously; while that form of inflammation which occurs after injury has generally a more favourable course. There have been cases of perfect recovery from cervical myelitis after a stab in the cervical region, and fracture and contusion of vertebræ; and this accords with the physiological fact that animals in which the cord was divided, so as to produce complete paralysis and anæsthesia below the seat of the lesion, have entirely regained the lost functions, perfect healing and regeneration of the injured organ having taken place. In such cases the reflex excitability which had been lost is seen to return; the condition of the bladder and the nutrition of the muscles improve; and ultimately common sensation and motor power are re-established. This healing process is generally protracted over six months, and sometimes even a longer period.

In other cases the condition of the patient remains stationary. The paralysis and anæsthesia remain nearly the same as they were at the end of the first fortnight; the symptoms on the part of the bladder and rectum vary, getting sometimes better and sometimes worse; the decubitus heals and breaks out again; and the patient, now an incurable invalid, may live for many months and even years, in a most uncomfortable condition.

Finally, myelitis may have become chronic, and yet be progressive in its character, so that from time to time fresh attacks occur which always leave the patient worse off than he was before. This progressive myelitis occurs in the descending or ascending variety. If descending, the grey substance

becomes more profoundly affected, as shown by loss of reflex excitability, and muscular atrophy and contractions; while decubitus and œdema may be rapidly developed. In ascending myelitis the principal new symptoms are dyspnœa, cyanosis, delirium, somnolence, and coma, which insensibly merges into the final dissolution.

CHAPTER VI.

EPILEPSY, HYSTERIA, AND CATALEPSY.

1. *Epilepsy.*

THIS most formidable of all diseases of the nervous system was, from its violent and terrible symptoms, and the reputed inefficacy of all remedies employed for its cure, by the ancients believed to be a direct and irrevocable infliction of Providence, and was therefore distinguished by the term 'Morbus sacer.' It is also known by the synonyms falling sickness, morbus divus, astralis, caducus, sorticus, lunaticus, herculeus, dæmoniacus, vitriolatus, comitialis, deificus, analepsia, apoplexia parva, St. Valentine's and St. John's disease, mal St. Jean, mal St. Gilles.

The following table shows the mortality from epilepsy in England and Wales during six periods of five years each, as well as the percentage of mortality, first of nervous diseases, and secondly of all diseases:—

Periods of five years	Deaths from Epilepsy	Percentage of Nervous Diseases	Percentage of all Diseases
1838–42	5,585	2·66	·32
1843–46	vacat	vacat	—
1847–51	8,667	3·62	·42
1852–56	10,339	4·01	·49
1857–61	11,689	4·31	·54
1862–66	12,359	4·21	·51
1867–71	12,290	3·98	·49
Total of thirty years	**60,929**	**3·86**	·47

It will be seen from this table that there has been a decided

increase in the mortality from epilepsy during the first four lustra which have been registered; while more recently it seems to have become less fatal, there having been a decided fall since 1862; but this fall has not been as great as the previous rise had been, so that the first lustrum is still much above the last.

The following table shows the

Influence of Sex on Epilepsy.

Periods of five years	Males	Percentage	Females	Percentage
1847–51	4479	1·86	4188	1·74
1852–56	5441	2·10	3998	1·55
1857–61	5972	2·41	5717	2·10
1862–66	6585	2·21	5774	1·96
1867–71	6483	2·10	5805	1·87

Men are therefore more liable than women to die from the falling sickness, the mean average for the former being 2·13, against 1·84 for the latter.

Age has likewise a decided influence on the mortality from this disease, which appears, from the annexed diagram G, to be very fatal in the first year, and altogether so in the first lustrum of life. At five the curve descends, rises slightly at ten, jumps up high at fifteen, and reaches the summit at twenty. At twenty-five it descends, and more considerably so at thirty. Another rise is perceptible at thirty-five and forty, and then a more decided fall occurs until fifty-five. At sixty it suddenly rises again, but after that the fall is steady, and at ninety the curve has descended to zero.

The influence of age on the occurrence of epilepsy in the two sexes is shown in diagram H. It is seen from this that the thick curve for males overlaps that for females in the first year of life, but that for the first period of five years both curves are nearly equal. From this until twenty-five the thin curve keeps at the top, but falls below the thick at thirty, and remains there until forty. After that a kind of zigzag movement is observed, there being no very decided prominence of one over the other, except between seventy and eighty, when the thick curve predominates.

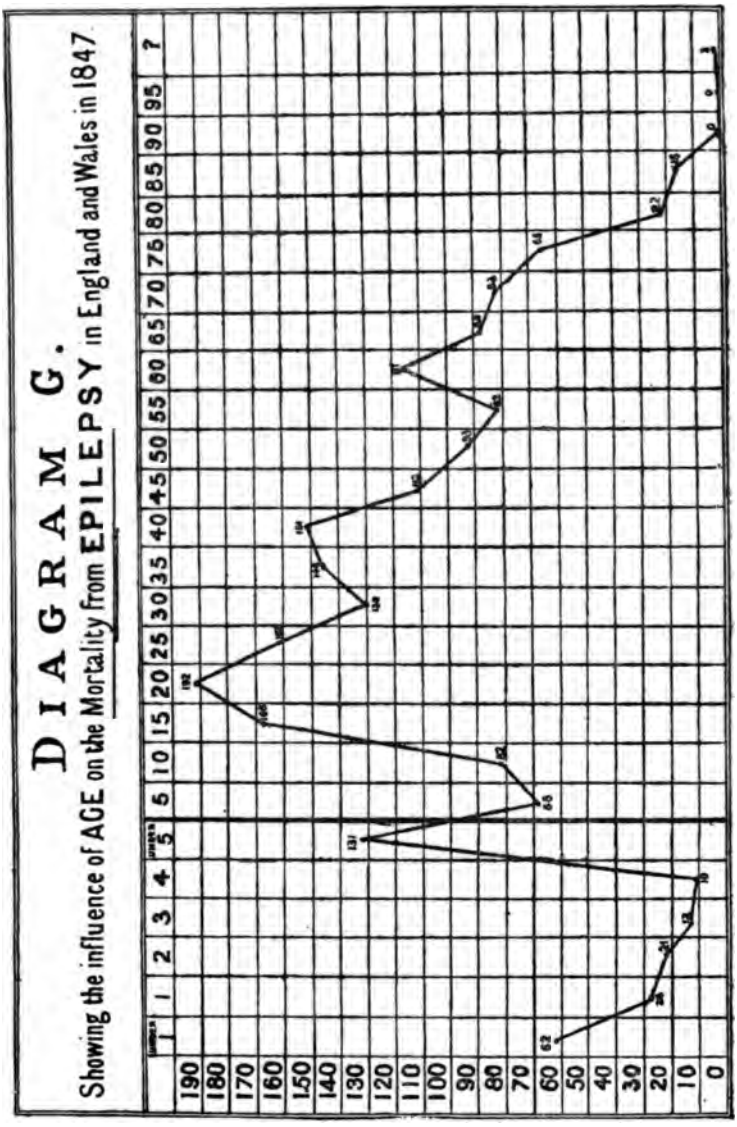
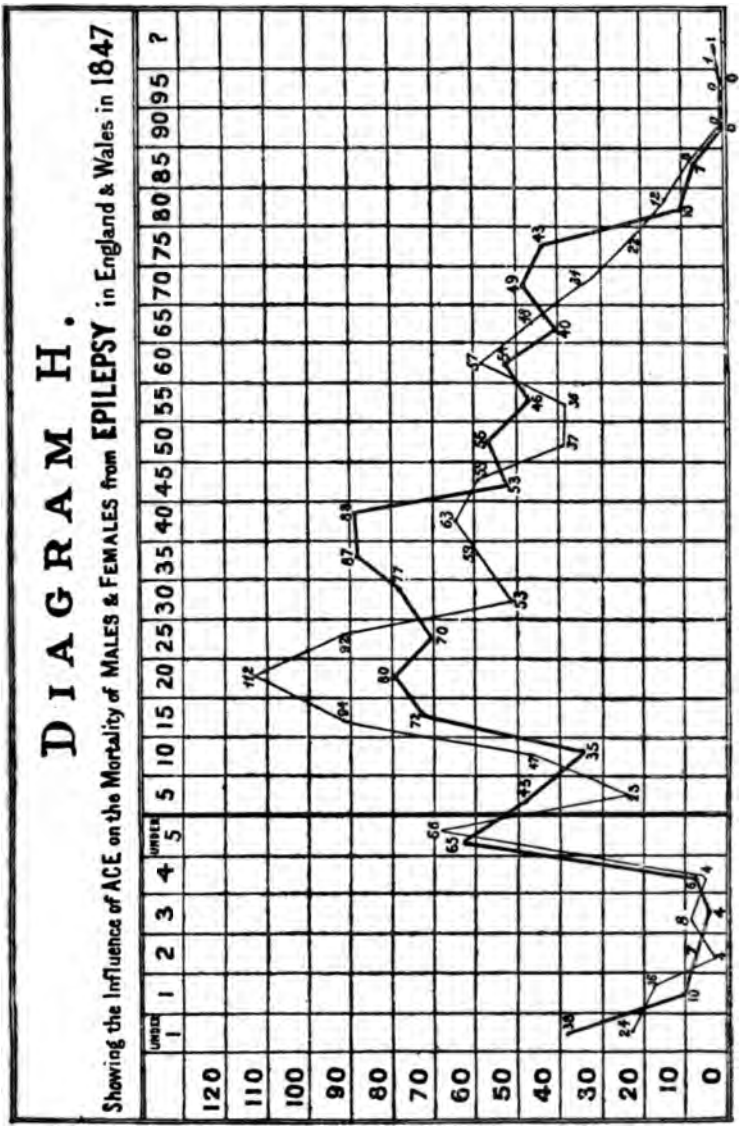


DIAGRAM G.
Showing the influence of AGE on the Mortality from EPILEPSY in England and Wales in 1847.
1 2 3 4 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 ?
190 180 170 160 150 140 130 120 110 100 90 80 70 60 50 40 30 20 10 0



The thin curve shows the deaths of females, and the thick curve those of males.

The principal divisions of epilepsy which have been current are *centric* and *eccentric*, *symptomatic*, *sympathetic*, and *idiopathic* epilepsy. Centric or idiopathic epilepsy, which results from a morbid condition of the pons and medulla oblongata, is, however, the only complaint which should be properly called epilepsy, and it is this which I mean whenever I employ the term. Authors speak of *eccentric* epilepsy where there is some palpable source of irritation outside of the nervous centres; such as disease of the kidney, stone in the bladder, worms in the intestines, uterine disturbances, etc. Now, it is quite true that under such conditions, epileptiform convulsions may be induced; but as a general rule these latter are merely reflex phenomena, arising from an irritation of the sympathetic nerves being transmitted to the nervous centres; and have only the outward manifestations in common with true, centric, or idiopathic epilepsy.

Epilepsy has been called *symptomatic* or *reflex* where convulsions arise from fracture of the skull, cerebral tumour, or spinal disease; where the patient is under the poisonous influence of alcohol, lead, or mercury; where there is a systemic cachexia, such as anæmia, struma, rickets, or syphilis; or where there has been injury to the extremities or peripheral nerves. The term *sympathetic* epilepsy is used where it appears to arise from irritation of the sentient or ganglionic nerves; such as neuralgia of the face, foreign bodies, insects or worms in the nostrils and frontal sinuses; the irritation of dentition; urethral and viscal calculus; indigestion; worms in the intestines; uterine and ovarian irritation, and masturbation. Some authors have assumed a further form of *vaso-motor epilepsy*, which they thought was caused by affection of the nerves of the arterioles accompanying the sympathetic nerve and its ramifications; as, for instance, after getting wet feet, etc. Dr. Brown-Séquard has described *spinal epilepsy*, which however consists in some cases of simple convulsions of the lower extremities, as seen in myelitis, and in others follows injury of the peripheral nerves. Some observers have gone so far as to deny that epilepsy is a real disease, and consider it only a symptom, the epileptic attacks being, in their opinion, invariably owing to some coarse structural disease affecting either the nervous system or remote parts. Such an opinion can only be attributed to insufficient observation

of the complaint, and will certainly never be endorsed by those who have studied the symptoms and habits of epileptics more particularly; for it is then seen that *epilepsy is a true general neurosis*, which, although principally located in the pons and medulla, affects the whole cerebro-spinal and sympathetic system of nerves, presents striking features in the sphere of mind, sensation, motion, and nutrition, and impresses upon the patients suffering from it a peculiar appearance, physiognomy, character, and habits of intellect. It is quite true that it may originate from peripheral or central irritation, as in Brown-Séquard's guinea-pigs; but the symptom becomes a disease when it has been reproduced several times, and may then be transmitted to the next generation. If, therefore, any distinction is to be made, it would be only justifiable to speak of primary and secondary epilepsy. *Primary epilepsy* would then be identical with the idiopathic or centric form of the disease, owing simply to a peculiar alteration in the pons and medulla oblongata, without any coarse structural alterations; while *secondary epilepsy* would comprise the reflex form, in which the first outbreak of the disease is owing to some definite irritation in the brain, spinal cord, or peripheral nerves. It should, however, be understood that both forms are identical in this way:—that although secondary epilepsy may originate in a remote lesion, it will not cease after the removal of the irritation, but that, when the characteristic alteration in the pons and medulla has once become established, fits will occur spontaneously.

That the pons and medulla oblongata are the principal seat of the disease is shown by the fact that epileptic attacks may still be caused in animals after removal of all other encephalic centres, while destruction of the two parts just named renders such attacks impossible. The spinal cord—which Marshall Hall thought the true epileptic centre, causing trachelismus and laryngismus, or tetanic convulsion of the muscles of the neck and larynx, with consequent asphyxia, loss of consciousness, and convulsions—serves only as a conductor between the medulla oblongata and the pons on the one hand, and the motor nerves and muscles on the other hand, but is of itself of no importance in the production of epileptic seizures.

We have already seen (p. 65) that cerebral anæmia suddenly

induced will cause all the symptoms of the epileptic attack, but although this anæmia unquestionably exists in the beginning of the fit, it is soon succeeded by venous congestion, in consequence of respiration being arrested though spasm of the glottis.

Pathological anatomy has as yet not helped us in explaining the nature of the peculiar condition of the brain which gives rise to epilepsy. In recent cases the post-mortem appearances are quite negative. Where, however, death takes place after a series of attacks, there are generally signs of venous hyperæmia in the nervous centres. The sinuses of the dura mater are gorged, the veins of the pia dilated, more particularly in the neighbourhood of the pons, medulla oblongata, and cerebellum; and there may be capillary hæmorrhage in the cineritious substance, as well as in other remote organs, such as the heart, lungs, mediastinum, and mesentery. In a few cases the heart has been found ruptured, but otherwise healthy, and this must have been owing either to spasm or to the violent efforts of the ventricles to squeeze the blood into the compressed arteries.

The changes which I have just mentioned have of course nothing to do with the causation of the epileptic attacks; and the peculiar alteration of the nervous system which causes epilepsy is as yet entirely unknown. Perhaps this alteration is only molecular; but be this as it may, it must be considered one of the great aims of the pathology of the future to elucidate this condition. The following is only a slight instalment of what remains to be done.

The *skull* has a peculiar shape in epileptics, more particularly where the disease has existed from childhood, or has come on in consequence of hereditary predisposition. There is a want of symmetry in the bones of the two sides; and they appear thickened and sclerosed, while in other cases they have been found attenuated. Stenosis of the foramen magnum and the spinal canal has also been discovered; and Virchow has drawn attention to the circumstance that stenosis of the skull is frequent in epileptics as well as in insane persons, and that in such cases there is a corresponding *aplasia of the brain*, which may be limited to a small region or spread over a considerable area.

Benedict,[1] who has recently given much attention to this matter, has found that in many habitually epileptic persons the tubera parietalia appear considerably displaced, the frontal tuberosity asymmetrical, and the forehead irregular. This want of symmetry is found to be most striking when a line is drawn and measured which extends from the root of the nose right across the skull to the spinous processes of the uppermost cervical vertebræ; as it is then seen that the frontal, parietal, or occipital part of one side is much less developed than that of the other, or that the frontal part is more developed on one side, and the occipital more than on the other. It may be assumed that under these circumstances a kind of compensation can be brought about, not only in the asymmetry of the skull but also in the corresponding aplasia of the brain. Provided such compensation be perfect, the irregularity may be devoid of pathological significance; but if imperfect, abnormal function of the brain may be the consequence. That aplasia and atrophy of nervous matter are often connected with convulsion and pain is well known; and I may here advert, as an instance of it, to the late Dr. Anstie's theory of neuralgia being caused by wasting of the posterior roots of the spinal nerves. The importance of these discoveries cannot be over-estimated, as predisposition, hereditary influence, and similar agencies may thus be explained. A different skull-shape, corresponding to a differently-developed brain, appears to give a more defined base for comprehending different functions than can be obtained if we seek refuge in generalities. The first glimpse is thereby afforded into certain well-marked anatomical differences, which would go far to render prognosis bad, if it was not also known that such irregularities may gradually disappear more or less under the influence of treatment, and that what was once a cause of disease, may come to be only a predisposing influence, which may remain latent under favourable conditions.

Benedict has also found that the condition known as oxycephaly is of frequent occurrence in epileptics. If the head be so fixed that the zygomatic arch is in a horizontal position, or so that the direction of the visual axes of both eyes is parallel, and

[1] 'Berliner Klinische Wochenschrift.' 1877. No. 32.

that the plane of these visual axes is parallel to the ground, it is found that the highest point of the forehead and the vertex have only a slight vertical difference from one another. In idiopathic epilepsy, on the contrary, this difference is very considerable, and is particularly noticeable when the highest point of the parietal bone is more forward in the region of the angulus bregmaticus. This shape of the skull is also found in habitual criminals, and more particularly in confirmed thieves.

The membranes of the brain have been found either normal, or thickened and adherent to each other.

With regard to the brain itself, the weight has been found increased by Echeverria,[1] and diminished by Meynert. The increase does not appear to have been owing to any greater development of nerve-cells, but to proliferation of the neuroglia. Want of symmetry between the two hemispheres, the left side being less developed than the right, has also been discovered. Meynert has met with atrophy of the cornu ammonis in several cases of epilepsy; other observers mention tumours, more especially of the cortex, syphilitic deposits, neuroma, sclerosis, softening, cancer, tubercle, and general wasting, with hydrocephalus, but it is evidently impossible that so great a variety of pathological lesions should have given rise to one and the same complaint. Most of the structural alterations which have been hitherto described have evidently been complications co-existing with, but foreign to, epilepsy itself.

The microscope has, up to the present time, assisted us very little in these matters. Schröder van der Kolck came to the conclusion that in the commencement of the disease there were no alterations of the nervous matter at all; but that after a time an intercellular albuminous effusion took place between the nervous fibres, causing sclerosis and fatty degeneration of the nervous matter and dilatation of the capillary vessels, with thickening of their coats. This ectasy of the capillaries was seen chiefly about the roots of the hypoglossus (in cases where epileptics had been in the habit of biting their tongue) and the pneumogastric nerves (where there had been no tongue-biting).

[1] On Epilepsy: Anatomo-Pathological and Clinical Notes. New York. 1870.

Van der Kolck, however, did not look upon these changes as causative of epilepsy, but as consequences of the hyperæmia developed by the attacks.

Echeverria has described the following as primary lesions of epilepsy:—capillary ectasy in the medulla oblongata, granular albuminous effusion, granular cells, abundant amylaceous bodies, not only in the neuroglia, but also in the transverse section of the floor of the fourth ventricle. The ganglion cells appeared pigmented, more especially in the nuclei of the hypoglossus and pneumogastric nerves. The same changes were found in the hemispheres, the centra ganglia, the cerebellum, and the cervical sympathetic nerve. In all these cases the connective tissue was found hyperplastic. Labimoff, however, has shown that the ganglionic cells are frequently found pigmented in the aged, without there having been any epilepsy, so that the importance of these alterations appears somewhat doubtful.

Ludwig Meyer, who has most carefully gone over the same ground, has come to the conclusion that all the vascular changes which have been described by various authors are secondary and not primary; and we agree with him in thinking that any uniform histological alterations causative of epilepsy have still to be discovered. All we know is that there is undue excitability of the ganglionic cells in the pons, where there is the central termination of the motor nerves; and on the floor of the fourth ventricle, where there are the grey nuclei for the motor cerebral nerves, as well as the vaso-motor and respiratory centre. These parts respond to stimulation more energetically than usual, so that instead of movements convulsions are produced, while the coma is a secondary phenomenon.

Dr. Todd was of opinion that the epileptic attack was owing to a morbid poison which gradually accumulated in the blood, and produced a state of undue excitability of the brain. This view is unquestionably right for the convulsions of uræmia, but no proof has been given that there is a poison producing epilepsy. It is true that carbonate of ammonia has been found in the urine several hours before the attack; and that after the attack is over, the perspiration and the expired air have been found to contain a considerable quantity of the same compound; but such changes are by no means constant.

We have in practice to distinguish three principal forms under which epilepsy presents itself, viz.:

1st. The true falling sickness, epilepsia gravior, haut mal, which is characterised by the sudden appearance of unconsciousness and general convulsions;

2nd. The milder form of epilepsy, epilepsia mitior, petit mal, the lesser evil, where there is only loss of consciousness, but no convulsions; and

3rd. Epileptic vertigo, which consists of irregular attacks of the disease, there being generally slight attacks of epilepsia gravior or mitior, with subsequent mental aberration and automatic action, for which the patient is not responsible.

A. *The Epileptic Fit.*

The true epileptic fit is generally preceded by premonitory symptoms, which may be remote or immediate.

Remote precursors are often found before the very first attack which takes place altogether, and are chiefly observed in children, and where the exciting cause of the complaint is fright. These precursors may indeed be looked upon as incomplete attacks. There is general tremor; pallor of the face, alternating sometimes with blushing; giddiness; a terrified appearance, which may be combined with excitement or depression; noises in the head; slight convulsive shocks in the hands and feet; and a severe epileptic fit may take place after this condition has lasted for a few hours, days, or even months.

What is called night-alarm, or 'night-mare,' in children, is often an incomplete epileptic attack. The children go to bed and to sleep in the usual manner, but after a time scream, endeavour to get out of bed, stare at some imaginary object, break out into a profuse perspiration, fall back exhausted and relaxed, and go to sleep again. Such attacks may be repeated several times in the night, and the little patients have no recollection of them on waking in the morning.

Those who have already had epileptic seizures may suffer in a similar manner when a fresh attack is threatening them. They feel depressed, tired, and excitable; are restless at night; the eyes are brilliant; there is a peculiar physiognomical expression, which

is easily recognised by those who have watched them in previous attacks; they complain of pressure on the head and palpitations of the heart. Or they have hallucinations, perceive a bad or a nice smell which is not perceived by anybody else; hear voices threatening them, cocks crowing, and cats howling; they see disagreeable faces, have terrifying dreams at night, and sexual excitement during the day.

The immediate precursor of the epileptic fit is called *aura*. This means originally a soft breeze; and the sensation described by some patients is, indeed, that of a hot or cold wind; but in general the aura may be defined as a sensation which seems to arise from a peripheral part of the body, and thence proceeds to the head.

The aura may be cerebral or mental, vaso-motor, motor, sensorial, sentient, secretory, visceral, or indefinite. The first of these is the one most frequently experienced, and characterised by sudden excitement, swimming in the head, seeing everything spinning round, and certain definite hallucinations. Children appear frightened and run to their mother, shouting, 'Mother, I am going to have a fit!' Occasionally there is a peculiar idea which the patient cannot describe when he is in his ordinary condition, but which is always suggested to him previous to an attack, and which is not only the last thing he perceives before the fit breaks out, but also gives him during that instant the certainty that he is going to have an attack.

The vaso-motor aura consists of a feeling of chilliness in the extremities, which gradually rises upwards. The parts become really cold, pallid, and anæsthetic, as from the prolonged influence of external cold, when all the blood seems to be withdrawn from the periphery by vaso-motor spasm.

The motor aura consists of convulsions in certain muscles or sets of muscles, which are mostly clonic and rarely tonic. They seem to arise chiefly from irritation of Hitzig and Ferrier's psycho-motor centres, as they have mostly a definite type, and resemble physiologically combined movements, only appearing in an exaggerated form. They may last for a few seconds or a few minutes. In a lady at present under my care, they occur in the right wrist (Jackson's regional spasm) almost immediately after lying down at night; they continue for two or three

minutes, can be stopped by pressure, and lead to an attack unless so stopped. I consider them owing to irritation of the ascending frontal convolution of the opposite side. Occasionally we meet with spasm of accommodation of vision, so that objects appear to become larger, or nearer, or further off.

The sensorial aura may proceed from all the different nerves of special sense. The most frequent aura appears to be a disagreeable smell, resembling tar, brandy, iodine, rancid fat, tainted fish, or high game; after that follow 'epilepsy of the retina,' viz., a sensation of coloured light or stars floating about, noises in the head, and a disagreeable sweetish taste in the mouth.

The sentient aura consists of a feeling of pins and needles which begins in the toes and fingers, and from there proceeds to the head; or there may be unilateral headache, and numbness in one side of the face.

The secretory aura is evidenced by profuse perspiration, lachrymation, and salivation; while the visceral aura consists of palpitations of the heart; a feeling of constriction, choking, pressure and heat in the epigastrium, retching, nausea, vomiting, intestinal colic, and tenesmus of the bladder and rectum. Sometimes there is only an indefinite sensation of malaise, and odd sensations which the patients find it impossible to describe.

An aura may be present without leading to an attack; and the latter may often be cut short when a sudden impression is made on the nervous system; as, for instance, tying a ligature round the arm or leg, shaking the patient, shouting to him, violently flexing the big toe, letting him smell ammonia, or giving him powerful medicines to swallow.

Is the nature of the aura peripheral or central? Some observers are inclined to look upon it as a peripheral sensation, while most consider it as simply an echo of a central condition. The aura appears to us part and parcel of the epileptic attack, and differing from the latter only in degree. Thus giddiness is a lesser degree of coma, and localised convulsions in the muscles of the wrist a slighter degree of general convulsions. We can only assume the aura to be peripheral in those cases where injury of peripheral nerves, such as the sciatic or the fifth, appears to give rise to epilepsy. The fact that an attack can

sometimes be prevented during the aura by a powerful impression being made upon certain peripheral parts, does not by itself speak for the peripheral nature of the aura; for under such circumstances there is no interruption to any peripheral excitement spreading towards the centre, but on the contrary reflex inhibition, spreading from the centre to the periphery. In a case of Odier's the aura consisted of convulsions of the brachial muscles, and could be stopped by ligature of the arm; yet at the necropsy a cortical lesion was discovered, which had to be looked upon as the starting-point of the aura as well as of the attack.

The epileptic fit consists of three periods, and generally commences with a scream, which used to be considered as a sign of pain, surprise, or terror of the patient, but is really owing to the spasm of the respiratory centre in the medulla oblongata, which causes a sudden convulsive action of the laryngeal muscles, by which the glottis is closed and a column of air expired. The face is at first pallid, but afterwards livid, and consciousness is lost. This is owing to irritation of the vaso-motor centre in the medulla, by which sudden anæmia of the face and the cerebral hemispheres is brought about. At the same time the patient goes down like a shot, whether he be standing or sitting, and there is only rarely time for him to lie down in a place of safety. The fall is not owing to loss of consciousness and sensibility, as has been supposed, but to the sudden convulsions of the muscles; for in that form of epilepsy which is called 'petit mal' there is also loss of consciousness, but the patient remains sitting or standing as quietly as before. If in the convulsive attack he goes down in the street, he may be run over; if in a room, he may fall into the fire, and seriously injure himself in a variety of other ways. Sometimes the skull is fractured, and death may result from purulent meningitis. The coma is, however, so deep during the attack that no pain whatever is experienced. The conjunctiva is quite insensible; the pupils are dilated and do not respond to the influence of light. The pulse is generally small, but sometimes quite unchanged; or the pulse of the radial artery may be imperceptible, while the heart's action is normal and the carotids are felt to throb violently. At the same time there is tonic rigidity of the whole frame;

the eyes are drawn backwards and upwards, so that the cornea is concealed under the upper eyelid. The face, and particularly the mouth, is fearfully distorted; the teeth are pressed against each other with great force, so that the tongue or the mucous membrane of the cheek is bitten. The head is drawn to the side, or forwards or backwards. One arm is generally drawn up and the other down. The body is in a state of opisthotonus, and respiration is arrested.

The pallor of the face, from anæmia of the blood-vessels, sometimes lasts through the entire first period of the fit. An ophthalmoscopic examination of the fundus of the eye is impossible under such circumstances, but it has occasionally been made just previous to an attack, when anæmia of the blood-vessels was observed. Generally, however, the pallor is quickly succeeded by lividity of the face, with swelling of the cheeks, lips, and tongue. This is owing to the closure of the glottis and the immobility of the chest-walls, which leads to accumulation of blood in the capillary vessels. The pressure on these is sometimes so great that they are ruptured, and effusions of blood take place. The whole surface of the body, but more especially the face, may thus become covered with petechiæ, looking like flea-bites. The complexion is sometimes quite coppery, and the patient looks like a red Indian. These petechiæ always show that the attack has been severe. In some cases they remain only for a few hours, but a degree of congestion may still be seen about the face for a few days after, and by means of a magnifying lens we may even then be able to distinguish traces of the petechiæ themselves. They are chiefly marked in the neighbourhood of the eyes and sometimes affect the conjunctiva. Capillary hæmorrhage, from excessive pressure, may, however, take place in other organs, such as the brain, and occasionally causes bleeding from the mouth and tongue where these parts have not been bitten.

In the *second period* of the attack, the insensibility continues, but the tetanus of the muscles is replaced by clonic convulsions, which consist of sharp, quick, short, and most violent muscular contractions separated by quiet intervals. They are sometimes so severe that the bones are fractured or dislocated, more especially the shoulder, elbow, and the lower jaw;

teeth are pulled out, muscles ruptured, and the tongue entirely bitten through. Nevertheless the occurrence of these clonic convulsions shows that the excitability of the pons and medulla is diminished. The convulsions are hardly ever quite bilateral; and when this occurs it should raise our suspicions whether the patient may not be simulating epilepsy. Sometimes there is tonic rigidity in one side, and clonic convulsions in the other, while in other cases the convulsions are strictly unilateral. They affect the flexors more than the extensor muscles. There is now foam at the mouth, which is often mixed with blood, either from the bitten tongue or cheek, or simply from rupture of the blood-vessels of the mucous membrane, causing extravasations, just as petechiæ of the skin are caused by rupture of the blood-vessels of the corium.

The face is more awfully distorted in the second than in the first period. Respiration is now forcible and accelerated, accompanied with râles, and the buccal and pharyngeal mucus is thrown backwards and forwards by the inspired and expired air. The patient now often begins to moan, and may do so for several minutes, which is not owing to pain, but to irritation of the corpora quadrigemina (p. 42). At the same time there are borborygmi in the intestines; flatus, fæces, urine, and even semen may be discharged, and projected to a considerable distance. There are now all the signs of venous hyperæmia, viz., swelling of the jugular veins, deep cyanosis, and prominence of the eyeballs. The pulse is now full and frequent, but occasionally very slow.

The *third period* is that of relaxation of the muscles; the clonic convulsions cease, either suddenly or gradually, the limbs become completely relaxed, and sometimes a kind of tremor is seen to run through the body. There is stertor and tracheal râles; the skin is bathed by a clammy perspiration, which frequently has an offensive smell, from containing carbonate of ammonia. The insensibility may continue for a few minutes longer, but generally consciousness now begins to return. The patient opens his eyes, and closes them again; he talks or murmurs in a dreamy fashion, and seems absent. The colour of the face becomes less livid, and the pupils begin to contract. Respiration is more tranquil and regular, the pulse continues full and

frequent. The ophthalmoscopic examination of the fundus of the eye shows considerable hyperæmia, which sometimes continues for twenty-four hours. The temperature is generally found normal or only slightly increased. The quantity of the urine is often increased; sugar, albumen, and spermatozoa have been found in the urine by some observers, but this is exceptional.

The entire duration of the epileptic fit is from two to ten or at most fifteen minutes. In the large majority of cases it lasts from three to five minutes. The first or tetanic period is generally the shortest, lasting from a few seconds to half a minute, or one minute, while that of the clonic convulsions is the longest, and lasts from three to six minutes.

As a rule the third period is followed by a deep and tranquil sleep, which lasts from one to several hours, and from which the patient awakens sometimes quite well. At other times there is utter want of recollection of what has happened; irritability of temper and great depression, or maniacal excitement, which may last for several days; or a state of confusion and imbecility combined with severe headache and soreness in the limbs. Sometimes aphasia and hemiplegia are discovered after the attack, but in such cases the latter was not one of true epilepsy, the convulsions having been owing to a coarse anatomical lesion, such as tumour of the brain, or cerebral hæmorrhage, or embolism of the sylvian artery.

Unilateral or bilateral anæsthesia and paræsthesia have also been observed after epileptic seizures, owing probably to capillary hæmorrhage at the time when venous congestion was at its acme. The temperature may rise to 99° or 99°·5, and continue so for a few hours.

The epileptic fit is often simulated for purposes of extortion, and on the Continent in order to escape military conscription. Epilepsy is more easily simulated than other diseases, as only an occasional performance is required, and the malingerer is after the exhibition at liberty to do what he likes. On the whole, however, it is not difficult to distinguish between real and simulated epilepsy. It is true that the malingerer may fall, scream, get red in the face, imitate the foam by chewing a bit of soap, lock his thumb into the hand, and pass his water and

fæces; but he cannot get his pupils dilated during the attack, and sensitive to light after it; he cannot make his temperature rise; he cannot get petechiæ in the face; and generally kicks both legs about in the same manner, while the true epileptic convulsion is more unilateral. The malingerer has no scars in the face, which are very frequently seen in the true epileptic; and he rarely has sufficient force of mind to bite his tongue.

The sphygmograph has also, in the hands of Voisin and others, furnished some interesting data. According to Voisin, the pulse becomes quicker shortly before the attack, has less force, and the sphygmographic elevations are lower and more approached to each other. During the attack, when the pulse is small, the undulations are slight, showing irritation of the vaso-motor nerves and contraction of the arteries. This, however, is soon succeeded by a paralytic condition of the sympathetic nerve, when the pulse becomes full and large, and the sphygmographic tracings higher and more pronounced. The convexity of the elevation points upwards. A few minutes after the attack is over, the elevations rise three or four times higher than before, make an acute angle, then descend and show dicrotism. Voisin explains these tracings by primary irritation and subsequent paralysis of the vaso-motor nerves.

Magnan has recently studied the state of circulation during the epileptic attack in dogs, in whom he induced convulsive seizures by the injection of absinthe, and then employed Ludwig's kymographion and Marey's polygraph. He found that during the first period of tonicity the pulse was accelerated and the arterial pressure increased; but that, as soon as the second period of clonic convulsions commenced, the pulse was greatly retarded, and arterial pressure very much diminished. With the beginning of the third period of muscular relaxation, the pulse regained its normal rate, or was slightly accelerated. All these phenomena, however, did not occur if the pneumogastric nerve was previously divided. Magnan concludes from these observations that during the period of tonicity the heart is in a state of partial tetanus, much the same as the other muscles, and cannot dilate properly, and that this state is brought about through the influence of the pneumogastric; while the retarded pulse and diminished arterial pressure of the second period are

due to temporary exhaustion of the cardiac muscle. It seems to us, however, better to explain them by assuming that there is at first irritation and afterwards paralysis of the sympathetic ganglia, the former causing a rapid, and the latter a slow pulse; for when the influence of the sympathetic system of nerves is removed, the inhibitory influence of the pneumogastric becomes paramount for the time being, with the result of cardiac action being retarded or even entirely suspended.

Death in the epileptic attack is rare, but it may occur if the patient is seized with a fit while bathing, when he may be drowned, or while eating, when he may choke. It is also occasionally consequent upon injuries received through the fall, but occurs only exceptionally from asphyxia or syncope.

Epileptic attacks may occur either both night and day, or only in the night, or only in the day; and in some patients they only come on at certain hours of the day. One of my hospital patients, a domestic servant, never had fits except the first thing in the morning, when she was lighting fires. Nocturnal attacks leave the patient less exhausted, both mentally and physically, than those which occur in the day-time. In women the menstrual period, including a few days before and after the discharge, is most exposed to fits; and in some women these come on only during cohabitation. In certain febrile diseases the attacks cease completely, and only reappear when the body-heat has fallen to its normal standard. Some patients have fits only once in two or three years, while others have a very large number in a single day. A patient whose case I have described elsewhere [1] had had over 10,000 attacks at the time she came under my care, and as many as 165 in one week; for several years past the minimum had been twenty in the week, not a single day having been free from them. Some months ago a boy was under my care at the hospital, who had between 40 and 50 attacks in the day, which makes about 1,350 in the month; and Delasiauve has described a case in which 2,500 fits occurred in one month.

Where severe attacks succeed each other very rapidly, a peculiar condition is produced which French observers have

[1] 'On Epilepsy, Hysteria and Ataxy.' London, 1866.

called *état de mal epileptique*, and which I propose to call by a shorter term, *epilepticism*. There is then no recovery of consciousness between the seizures, which follow each other like thunderclaps; the respiration is much accelerated; the pulse rises to 140 or 160 beats in the minute, and is excessively small; the temperature ascends to 106° and 107°; there is deep stupor and collapse; and the patient dies after two or three days, without having recovered his consciousness. Epilepticism is, however, not invariably fatal, and the most reliable sign of improvement in the patient's condition is a lower temperature.

The same person generally has the same attack. One person gives the same initial scream, falls forwards, the tongue is bitten in the same place, and the convulsions last exactly five minutes. Another patient never screams, nor bites the tongue, but falls towards the left side, foams much at the mouth, and passes his water in the attack. There are thus infinite varieties of the attack, and the classical type of it which I have just described may undergo numerous modifications.

B. *The Lesser Evil.*

The second form of epilepsy is the *petit mal*, the *lesser evil*, *epilepsia mitior*. This consists of loss of consciousness without convulsions; is preceded by vertigo, or a strange sensation rising from the epigastrium to the head, and followed by confusion. The loss of consciousness is generally short, that is, a few seconds or a minute; but in exceptional cases it is prolonged to half an hour or more. The patient does not fall, but remains sitting or standing; he looks confused and astonished, and if he holds anything in his hand at the time he drops it. The loss of consciousness in these attacks is so complete that no pain whatever is experienced. A patient of mine, a retired merchant, aged 47, once had an attack of 'petit mal' while standing with his back to the fire. He remained standing, but it is supposed that he leant heavily against the chimney-piece, and that it was in this way that his clothes caught fire. However this may be, he was found, still standing erect and unconscious, leaning against the mantle-shelf, with his clothes all burnt to cinders and lying about the carpet in charred bits. On his back there was a deep

burn as large as the hand, and he must have remained exposed to the fire for a considerable time, yet had not felt anything at all of the burn as it proceeded to consume his flesh.

C. *Epileptic Vertigo.*

This is one of the most singular manifestations of epilepsy, and possesses not only great pathological but also considerable medico-legal interest. It is not yet known throughout the profession that patients, while in this condition, perform automatic acts which may be perfectly innocent and harmless, but which may also be criminal; and it is my firm conviction that they are absolutely irresponsible for anything they may do while thus affected. The subject of epileptic vertigo attracted some time ago considerable public attention, on account of a peculiarly unprovoked murder having been committed by an epileptic while in this state; and as it is still comparatively new and only little explored, I subjoin the particulars of a number of cases of it, which have been under my care in private and hospital practice.

A married lady, aged 28, childless, had her first fit on her wedding day. She continued afterwards to have regular epileptic attacks with convulsions, and simple losses of consciousness. In one of these latter, which lasted about five minutes, she took a set of false teeth, which she wears, out of her mouth, and washed it in a tumbler, without knowing anything at all about it when she awoke.

An unmarried lady, aged 24, fell on the ice when fifteen years of age. At that time she had her catamenia, which were suddenly arrested, and she had her first fit the day afterwards. Ever since she has been subject to nocturnal epilepsy, which at first selected the time of menstruation, but afterwards showed itself at irregular intervals. She gives a scream, bites the tongue, foams at the mouth, and has convulsions which last about four minutes. As soon as the attack is over, she gets up, lights a candle, and wants to get out of her bedroom window. This desire lasts for about half an hour. A maid is always with her to see that she is safe. She is quite unconscious during this time; but on one occasion, when her mother sharply told her to go to bed again, she did it. Her mother then left the room,

upon which the patient said: 'Now I may get up again!' I may add that there is not the slightest symptom of hysteria in this case, and that there is perfect unconsciousness while the tendency to somnambulism lasts.

A French polisher, aged 40, has for some time past had the following attacks:—He suddenly feels a severe pain at the back of the head, and 'a thrilling sensation seems to run through him as if he were about to die.' Or 'a vapour seems to rise on his brain and muddles him,' and he then entirely loses his consciousness. While in this condition he generally does something odd; for instance, when at dinner, scratches the plate with the knife, or tears up paper, or his clothes, or pulls a handkerchief over his head. If in the street, he puts mud on his clothes. When he comes out of these attacks, he feels very confused, and sees double for two or three minutes. He recovers himself generally in an hour or two, and has such fits two or three times a week.

Another patient, who was subject to great convulsive seizures, described his attacks of vertigo as follows:—On reading aloud from a newspaper, he suddenly stops, being unable to proceed any further; he then remembers nothing, but wakens up in four or five minutes, finds the paper crushed into a very small compass between both hands, and then goes on reading, but does not commence at the place where he stopped, and is not aware of what has happened. He has never committed any acts of violence while in this condition; but on one occasion, when his mother was near, he seized one of her hands between his two, and held it 'as with a death grasp' the whole time that the fit lasted. Another time he crushed his hands between the iron rails of his bed, and found that the nails of the first and second fingers of the left hand had been torn from the quick. A common occurrence with him is that, when he is walking in town or country, he is suddenly, as it were, 'wakened up'; he finds that he has been unconscious, but that he has gone the 'correct road,' and left nothing behind. Once, however, he found that he had thrown away his pocket-handkerchief while in this condition. If in town, he sometimes 'wakens up' in some shop, just on the point of being 'put out.' He once went to his bank to pay in some money shortly after a fit; and

after having put a sum down for the clerk to count it, he took four sovereigns back, and put them into his waistcoat pocket. When the clerk told him that he must alter the entry, as he had taken four sovereigns back, he stoutly denied it. The clerk then requested him to feel in his waistcoat pocket, and 'sure enough, the money was there.'

Another patient has, after slight epileptic seizures, attacks of vertigo, in which, although utterly unconscious, he goes on doing what he happened to do before. For instance, he may, when at dinner, suddenly lose himself while eating from the joint, then eat pastry and cheese, and get up from the dinner table without knowing anything whatever about it. At times he is quite silent when in this condition; but at other times he will talk incoherently—for instance, tell his brother on a Sunday, 'Well, turn up the card; what's trumps?' The same patient, being very fond of playing at billiards, has gone into a public billiard-room, and, during one of these attacks, taken away the cue from the player and begun to play, without being in the game. On another occasion, while walking in the City, he suddenly lost himself, ran up a scaffolding, and began to shout that he wanted to knock the Queen and the Prince of Wales down. He became so violent that he had to be taken to the workhouse infirmary, and when awakening there had no recollection whatever of what had taken place. In this case the epilepsy came on from drinking a large quantity of new rum when the patient was out in the West Indies.

A young lady, aged 21, who is now under my care, fell from a great height when a child, which produced concussion of the brain. She had to be in a dark room for two or three years, and has always remained rather childish. When she was 20, attacks of vertigo came on, chiefly about the time of the period, in which she suddenly gets up and stands against the wall, repeating all the time, 'Oh, yes! oh, yes! oh, yes!' This will sometimes go on for a whole afternoon. She is perfectly unconscious all the time, and does not hear anything when spoken to; nor is she at all aware that she is subject to such attacks.

A clerk, aged 29, has losses of consciousness which last baout five minutes. They take him anywhere; and he is par-

ticularly distressed about the matter, because while in this state he will unbutton his trousers and pass his water, wherever he may be. He has done this in a crowded thoroughfare, and in his office; and he has several times 'got into trouble' in consequence.

The transition from such states as I have just described to those of *epileptic mania*, in which the patient commits suicide, homicide, rape, and arson, is easy and natural. Some observers are inclined to think that epileptics may suddenly become maniacal without having had an epileptic attack, the mania taking the place of the fit; but from what we have seen in practice, we are inclined to the opinion that vertigo, as well as mania, always occur after attacks, and more particularly when these have been slight. During the fit there is a severe discharge or explosion, which affects not only the pons and medulla, but also the cineritious structure of the hemispheres. While, however, the pons and medulla recover more or less rapidly, the higher nervous centres, more particularly those representing the intellect and the moral control, remain in a state of exhaustion, or at least reduction; and what takes place after such an attack is due to the automatic action of the lower centres, which are healthy, except that they are for the time being deprived of the control of the higher centres. Under these circumstances an epileptic may be capable of very elaborate actions; and it is a general rule, that the slighter the fit the more elaborate and the more full of apparent intention are the actions which follow.

Epileptic mania is only a severe degree of vertigo, characterised by furious excitement. The face has an expression of rage; the breath an ammoniacal smell. The patient destroys anything which comes in his way, spits into peoples' faces, stamps with his feet, knocks the first comer down, and then suddenly becomes perfectly quiet, and has no recollection of what he has done just before. A man in this state may commit atrocious murders, rape, arson, and robbery. Sometimes the patient feels the attack of mania coming on, and warns the bystanders that he is going to do something dreadful.

Griesinger has described a class of cases to which he has given the name of *epileptoid*. Such patients suffer from

megrim, vertigo, dyspepsia, syncope, hallucinations, and all kinds of abnormal sensations. These symptoms come on periodically and paroxysmally, and occur in persons who have a hereditary neuropathic disposition. The close relation of some of these conditions with epilepsy cannot be questioned; yet Griesinger seems to us to have gone somewhat too far, and to have drawn more things into the epileptic vortex than is really justifiable.

Even where there is no epileptic vertigo and automatic actions of the lower nervous centres, the mental condition of epileptics is almost always peculiar. They are not really insane, but eccentric, suspicious, ill-tempered, perverse, fretful, and difficult to get on with. They seem to expand only with those who are similarly afflicted, but are otherwise shy, peevish, gloomy, and exclusive. Their intellect is below the average, and their memory impaired, more especially after attacks. Their judgment is often incorrect, and they are generally unhappy. This, however, is in a great measure owing to the circumstance that most careers which are open to other people are closed to them; they generally lose their situations as soon as they are known to have fits, and are looked upon with dread and dislike. They are fond of remaining an indefinite time in hospitals, where they amuse themselves with playing at cards and reading the papers, without showing any desire for real work; yet, on appealing to their better instincts, we may often encourage them to acts of real devotion.

Dr. Radcliffe has always found deficient circulation in epileptics; but such is not my experience. Epilepsy occurs in extremely powerful men, with a splendid circulation, and whose health is, in every respect but this, perfect. Indeed, some epileptics are perfectly well between their attacks, and look the very picture of health, even where the disease has lasted a very considerable time. It is related that Cæsar, Petrarca, and Napoleon I. were subject to epileptic fits; and these men could not have accomplished what they have done unless their general health had as a rule been satisfactory.

Amongst the causes of epilepsy, the most important is hereditary predisposition. Brown-Séquard found that the young of guinea-pigs, which had been artificially rendered epileptic,

became subject to attacks without having been operated upon themselves. Echeverria could trace the hereditary influence in 80 out of 306 cases; and I have reason to believe that it is even more frequent. Such an influence is often denied, as it seems to reflect injuriously on the family; and it is often shown to be present only when the history of the patient becomes better known. It occasionally overlaps a generation, and appears in the next. Moreover, it is not actually necessary that there should have been true epileptic seizures in preceding generations; for chorea, insanity, severe forms of neuralgia, and more particularly pronounced hysteria of the mother, are of great influence in the production of epilepsy in the offspring. Where epilepsy is hereditary, the infant is liable to attacks of eclampsia, and true epilepsy is developed about, or at any time previous to, puberty. If the twentieth year is passed without the occurrence of epileptic attacks, the hereditary predisposition may generally be considered as having worn itself out in that particular instance.

The next cause is alcohol. Magnan has produced epileptic attacks in dogs by injecting absynthe into their veins; and the drunkard is frequently attacked by epilepsy. The first fit often takes place during alcoholic intoxication; and hospital experience has shown me that by far the largest number of fits amongst the lower orders in London takes place on Saturday night or early on Sunday morning, after a prolonged stay at the public-house. Excessive mental work, grief, and anxiety come next; but masturbation and sexual excesses are not so important in this respect as they were formerly believed to be. Bad nutrition from want of food, hæmorrhage, and exhausting diseases seem to produce epilepsy only occasionally. The influence of syphilis in this respect will be discussed in the last chapter of this volume. The temperament of the individual has a considerable influence. Epilepsy is more likely to become developed in persons who are very impressionable, excitable, and show the neuropathic disposition in a general way.

Structural lesions of peripheral nerves may likewise cause this disease. In such cases there are generally weeks or months between the infliction of the injury and the first attack; but during the intervening period there is often pain and convul-

sion in the area of the injured nerve. If an attack ultimately takes place, it is preceded by an aura in the sphere of the affected nerve. This kind of epilepsy has been termed reflex epilepsy, as it appears to be owing to a continued morbid stimulation proceeding from the periphery to the centre, and ultimately causing convulsibility of the pons and medulla. The nerves in the sphere of which this occurs more particularly are the sciatic and the fifth, and the lesion is generally laceration of the nerve and less pressure from tumours, such as neuroma.

Injury to the skull and to the cortex of the brain by fragments of bone may have the same effect. Circumscribed lesions of the cineritious substance cause that form of 'cortical epilepsy' which Dr. H. Jackson has studied with so much attention.

The exciting causes of the first attack are extremely numerous; but it must be remembered that these causes are unable to produce real epileptic fits, unless the peculiar alteration in the pons and medulla which causes the epileptic condition was pre-existent in a latent form. Amongst these exciting causes the most frequent are fright, grief, rage, terror, disgust, and excessive joy; the first appearance of the catamenia; the accomplishment of marriage; over-eating and excessive drinking, especially after prolonged abstinence; over-exertion; severe diarrhœa; and convalescence from acute diseases. The favourite time for the outbreak of the complaint is the second dentition, puberty, and the climacteric period.

2. *Hysteria.*

This disease has been known from the commencement of civilisation, and was so called by the physicians of Ancient Greece, who believed it to arise from the freaks and vagaries of a dissatisfied and ill-tempered uterus (*ὑστέρα*). Plato and his followers described this organ as an animal endowed with spontaneous sensation and motion lodged in another being, and ardently desirous of procreating children. If (argued these philosophers) it remained sterile long after puberty, it became indignant at its unnatural condition, travelled through the

whole system, arrested respiration, and threw the body into extreme danger, until it became pregnant, whereby its wrath was appeased, and it behaved well ever afterwards.

Pressure of the uterus upon the various organs of the body was considered to be the mainspring of all the sufferings of hysterical patients. Where there was a feeling of suffocation, it was due to the uterus compressing the throat and the bronchial tubes; coma and lethargy in hysterical women proceeded from the womb squeezing the blood-vessels which go to the brain; palpitations arose from the uterus worrying the heart; and if there were a feeling of pain and constriction in the epigastrium, it was again the womb engaged in a relentless attack on the liver. Even so recent a writer as M. Landouzy has endeavoured to prove that the sick or dissatisfied uterus is the only source of hysteria.

A more accurate observation of facts, and a less prejudiced interpretation of the same, has gradually led us to different views on this subject. The credit of having upset the uterine theory of hysteria belongs chiefly to M. Briquet, who was the first to apply the numerical method to this branch of pathological enquiry, whilst at the same time carefully guarding against the dangers which beset the path of statistical investigation. Romberg first directed attention to the fact that reflex excitability is largely increased in hysteria; but he did not lay sufficient stress on the emotional character of the disease.

And yet it is this which serves to explain not only the infinite variety of symptoms, but also the causation and progress of hysteria. The multitude and apparent incongruity of its symptoms have perplexed and bewildered those observers who were without this clue to the comprehension of their nature. Rivière called hysteria not a simple but a thousandfold disease. Sydenham asserted that the forms of Proteus and the colours of the chameleon were not more various than the divers aspects under which hysteria presented itself; and Hofmann said that hysteria was not a disease, but a host of diseases. Yet all the symptoms, such as convulsive attacks, fainting fits, pain, cough, difficulty of deglutition, vomiting, borborygmi, asthma, hiccough, palpitations of the heart, tenesmus of the bladder,

general and partial loss of power, catalepsy, coma, delirium, etc., flow from the same source; and may be classified as functional spasms and paralysis, anæsthesia and hyperæsthesia, resulting from painful impressions, whether mental or physical, which act on the emotional centres of the brain.

Being guided by this fundamental principle, the transition from physiological to pathological manifestations is easy and natural. All symptoms of hysteria have their prototype in those vital actions by which fright, grief, terror, disappointment, and other painful emotions and affections, are manifested under ordinary circumstances, and which become signs of hysteria as soon as they attain an undue degree of intensity.

If an impressionable woman, who is naturally somewhat devoid of self-control, or who may have lost this faculty from being worn out by disease or anxiety, is suddenly told that the house is on fire, or that she has lost a near relative, the following symptoms are generally produced:—She has a feeling of constriction in the epigastrium, oppression on the chest, and palpitations of the heart; a lump seems to rise in her throat and to choke her; she loses the power over her legs, so that she is for the moment unable to move; and she wrings the hands in a spasmodic manner. If these symptoms become intensified, the well-known signs of hysteria are developed, which I have just classified under the four heads of functional spasm and paralysis, anæsthesia and hyperæsthesia, and which result from painful impressions being transmitted to the emotional centres of the brain.

The nature of the peculiar constitution predisposing to hysteria has to this day been a matter of controversy. Hippocrates believed that women who had an abundance of 'seminal fluid,' and who suffered from leucorrhœa—that is, the lymphatic and the pale—were liable to become hysterical; while Galen held that the strong, the fleshy, the sanguine women had a greater tendency to it. Subsequent writers have sided sometimes with Hippocrates and sometimes with Galen, but mostly with the latter. There is, however, no peculiar *physical* constitution predisposing to hysteria, since the disease indiscriminately invades women of all kinds of such constitutions. Nor has the intellect anything to do with it; for some hysterical women are very clever,

while others are the reverse; it is rather the mental and moral peculiarity which exercises an all-powerful influence on the production of this disease.

Women whose sensibility is blunt never become hysterical; while those who are readily accessible to impressions, and who do not possess much volitional energy or force of character, who feel acutely and are liable to strong emotions, without being able to control themselves, are certain to become hysterical if made to suffer mental agony or prolonged physical pain. This high degree of sensibility is not confined to any particular rank of society, but may be found equally strong amongst the lower classes as with the upper ten thousand. Hysteria, therefore, occurs in women of all ranks and orders. It is frequent in the higher classes of society, in ladies who lead an artificial life, who do nothing, whose every wish or whim is often gratified as soon as formed, and who are very apt to go into hysterics at the slightest provocation or contrariety. For them, real honest work, the pursuit of an object in life, such as the education of children or some charitable undertaking, is often the best cure. Again, we find plenty of irritable and impressionable women in the lower classes; and as want, grief, and anxiety are common amongst them, they are very prone to hysteria. Such women are sometimes cured by an improvement in their social position.

As emotion and anxiety on the one hand, and highly impressionable women on the other hand, are found in all inhabited quarters of the globe, hysteria is not confined to any particular climate or country. The common belief that this disorder is more frequent in tropical than in temperate or cold climates is not founded on fact, for we find hysteria not only in the South, but in the highest latitudes. The Russian ladies are particularly hysterical. The same is the case with the Swedish, Polish, and Swiss; and hysterical women are even found amongst the Esquimaux and Greenlanders, and in Iceland. On the other hand, there is no doubt that the circumstance whether women live in towns or in the country is of considerable influence in the production of the disease. Although hysteria does occur amongst rustics, yet it is far more frequent in large towns, where the emotions are keener and more apt to be played upon

than in the country; and the Latin races are far more susceptible to it than the Anglo-Saxon.

Similar considerations serve to explain the influence of sex in the production of hysteria. Those observers who believed the sick or dissatisfied uterus to be at the bottom of all the mischief, were obliged to maintain that hysteria occurred only in women. And it is certainly infinitely more frequent in females than males, although not on account of the uterus, but by reason of the higher degree of sensibility generally possessed by women. Yet it does occur in males as well as in females, if they are highly sensitive and subject to painful emotions.

As cases of hysteria in male patients are rare, I will give the particulars of a case in point, which occurred some time ago in my practice.

In July, 1868, I was called to see a foreign gentleman, aged 23, who had, as I was informed, been suffering from 'brain fever' during the last three days. His friends gave me the following history:—About a week ago he received a letter informing him that a young lady to whom he was attached had engaged herself to another gentleman. On receipt of this news he flew into a violent rage, and smashed a good deal of glass and china which happened to be on the table. He then began to talk about the frailty of women, indulging in most sarcastic remarks on the sex in general. Two days afterwards a friend took him to Richmond for a change. The whole of that day he was silent and morose, and returned in the evening in a very bad humour. During the night he again became violent, and broke several pieces of furniture. Next morning, about nine o'clock, he had a severe convulsive attack, in which he bit his tongue and foamed at the mouth, the limbs being considerably distorted for about fifteen minutes consecutively. After the attack he was exhausted, and remained so for about a quarter of an hour, when a similar paroxysm occurred, which was followed by many others in the course of the day In all these attacks consciousness was not entirely lost. Between the fits he would either lie quietly in bed, or suddenly jump up, roll about the floor, and try to strangle himself, or to smash furniture. A medical practitioner was called in, who prescribed five grains of bromide of potassium several times daily. On an

attempt being made to give him this medicine in a wineglass, he ground the glass to pieces between his teeth, and it was believed that he had swallowed some pieces of it. I saw him the day after, at five o'clock p.m., when I found him in much the same condition as just described—viz., occasionally seized by convulsive attacks, half-conscious, and violent. He would not answer any questions. There was evidently no meningitis, as the pulse, the skin, and the pupils were in their normal condition. Under these circumstances, I looked upon the case as one of acute hysteria, caused by sudden and severe mental emotion. As it was impossible to make the patient swallow any medicine, I rapidly injected, during a temporary lull in his strugglings, half a grain of acetate of morphia into the cellular tissue of the forearm. Five minutes afterwards he was perfectly composed. In fifteen minutes he went to sleep, and slept for five hours consecutively. He then awoke, and was violently sick several times, although he had eaten nothing for two days. He got out of bed next morning about ten a.m., and was quite calm, making no allusion to what had occurred. I saw him again at five p.m., and as there were then slight symptoms of restlessness returning, I repeated the injection, but used only one-sixth of a grain, the patient not offering the least resistance. The next day I was informed that he had had a good night's rest, and was perfectly rational and collected. I saw him again (not professionally) six weeks afterwards, when, in answer to my inquiries, he said that he was quite well, and never alluded to his having been ill. I heard subsequently from his friends that he had continued in excellent health ever since, and was engaged to be married, having quite dismissed from his mind all thoughts of his former attachment.

I will only add that I generally, in cases which require the subcutaneous administration of sedatives, use one part of atropia to twelve parts of morphia, in order to counteract the sickness and vomiting; but in the case just recorded, I thought it best to utilise the emetic effects of morphia simultaneously with its sedative action.

The common belief is, that hysteria does not occur in childhood and advanced age; but this is erroneous, for amongst 820 well-marked cases of hysteria which I have collected from

medical literature, there were 71 patients under ten years, and 28 over the age of forty-five. Hysteria is, therefore, not confined to the period of puberty, as the advocates of the uterine theory would have it. During childhood, the female sexual organs are in a state of perfect repose, and do not give rise to sufferings; but nervous sensibility is high, and reason still dormant; so that, if painful emotions be frequently repeated or be unusually powerful, we have all the necessary conditions for the development of the disease. In accordance with this view, we find that when children are hysterical, the cause is almost always maltreatment by the parents (especially stepmothers) or nurses, and excessive sensibility inherited from the parents.

In accordance with this we find that the chief cause of hysteria is hereditary predisposition; hysterical parents beget hysterical children. Chorea, epilepsy, insanity, delirium tremens, and a generally increased excitability and convulsibility, *i.e.*, the neuropathic disposition on the part of the parents, have, however, the same influence as the actual existence of hysteria itself.

Nevertheless it is found in practice that the education of children, and more particularly of girls, may considerably modify the original tendency. Where there are germs of hysteria in a child, they may be destroyed by firmness combined with kindness on the part of the parents, and particularly of the mother; while, on the other hand, a foolish or bad-tempered mother may foster a slight predisposition, so that it develops into rampant hysteria. Two extremes have to be guarded against in education, viz., 1st, unreasoning kindness which yields to every whim, and renders the child capricious and unhappy; and 2nd, too great sternness, which must cause moral shocks to an imaginative girl. Coddling is therefore as bad as excessive hardening. But probably the worst influence is that of a capricious education, where the mother is apt to run from one extreme to another, and the child never knows what to expect, so that the mind becomes completely unsettled. The circumstance that the education of boys is generally entrusted to masters, explains, apart from the influence of sex, why hysteria is rare in males.

The hysterical mother also develops the germs of hysteria in

her daughters by her own example. This is more especially the case where convulsive fits take place habitually. There exists a nervous contagium as well as a physical one, and it acts in an equally subtle manner, selecting some and sparing others, but it is frequently found that the nurses of epileptic or hysterical persons become ultimately themselves hysterical; and epidemics of hysteria in hospitals, convents, ladies' schools, manufactories, and prisons for females, are for this reason of frequent occurrence.

Between 15 and 20 years of age, hysteria is most frequent, in consequence of the radical change which the nervous system undergoes during that period. Within those years girls begin, as it were, a new existence; they leave the nursery and its habits, and, imagination reigning supreme, they enter upon the world with its passions, troubles, and disappointments; and if painful emotions be frequently and powerfully experienced, hysteria is the inevitable result, provided the system is predisposed for it. After 20, the disease becomes more rare—a circumstance which cannot possibly be explained by the uterine theory; for at no other age are the female sexual organs subject to more considerable disturbances than after that time of life. The condition of the nervous centres, however, gives us a satisfactory clue to this circumstance. As imagination gives way to a more matter-of-fact view of life and the world, illusions vanish, and impressions and sensations are kept more under control than previously. As age advances, hysteria is still more seldom met with, because the mind has become settled and critical, and is less accessible to sudden impressions and emotions.

Anæmia, either primary or after hæmorrhage, a generally defective state of nutrition, convalescence from acute diseases, phthisis, and certain diseases of the sexual organs, may act as exciting causes of hysteria where there is the nervous predisposition already in existence. Amongst the latter diseases it is chiefly flexions and chronic inflammation of the womb and ovaries which are of influence in this respect, while cancer or other severe maladies do not seem to lead to the outbreak of hysteria. The reason for this is probably that cancer only appears at a later period of life, when there is but little consti-

tutional tendency to the production of hysteria. Enforced abstinence or excessive indulgence in the sexual appetite may also act as an exciting cause; and hysteria is generally more intense during menstruation, pregnancy, and lactation. Yet in a very large number of hysterical women the sexual functions are in every way in perfect order, and the disease is not in any way influenced by the events in the sexual life of the patient.

Hysteria is almost always a chronic disease, the symptoms of which are developed in a regular manner. At first the complexion becomes pale and sallow, the skin dry and hard, the patient loses flesh, and complains of headache. The appetite is fanciful and feeble; some patients having great dislike to anything but water, vinegar, and confectionary. Biliary secretion is tardy, and constipation habitual. Many hysterical women only go to stool once or twice a week, and in exceptional cases constipation may last for a fortnight or three weeks. There is generally a large accumulation of gas in the intestines, giving rise to colics or borborygmi. The epigastrium is very tender to touch, and pain in this region is much complained of; it is worse after emotions or walking, but not after meals. There is also pain at the level of the middle part of the left false ribs and the left side of the spine. Abdominal pulsation is frequent, and menstruation generally troublesome. The blood is often impoverished, the pulse being quick, small, and feeble. The temper is irritable. The smallest contrarieties of daily life which have scarcely any effect on other people, are sufficient to annoy or to upset the patient. A trifling variation in the day's temperature, a shower of rain, a change in the wind, a somewhat prolonged walk, and any little disappointments, are sufficient to make her thoroughly miserable. All of a sudden, however, things take a turn, and she is charmed with everything, more particularly so with a new doctor. Yet as a number of small miseries have to be encountered in life, the temper is as a rule bad, and the character capricious. There is great aversion to certain animals, such as spiders, frogs, and mice, the mere sight of which will cause fainting and convulsive attacks; while on the other hand, intense devotion to birds and cats is displayed. A tendency to lie, cheat, and intrigue is

developed; and other people's misfortunes are enjoyed. The imagination sometimes takes a decidedly erotic turn, more especially in those whose appearance is not attractive to the other sex. In order to make themselves interesting, hysterical girls and women will swallow pins, starve themselves, attempt suicide, drink urine and eat fæces. They have been known to put frogs into the rectum and the vagina, and pretended not to have gone to stool for months or even years. The Welsh fasting girl and Louise Lateau, the stigmatist, are well-known examples of this condition. On the other hand, if we succeed in rousing their enthusiasm they are often capable of intense devotion and exertions—for instance, in nursing the sick and wounded in time of war.

Hallucinations and delirium occur in the severer forms of the disease. The patient then has frequent and intimate intercourse with the saints and the Virgin Mary, while the Evil One generally lurks in the background. Religious hyperæsthesia is usually combined with erotic tendencies. Stories of rape are sometimes invented, and perfectly innocent men have been put into prison on such false accusations. Sometimes even a murder is committed, and suspicion most ingeniously diverted from the perpetrator to somebody else. The story of Constance Kent, who murdered her brother at Road, and made it appear that her father was the murderer, is an illustration of this condition. Somnambulism and the phenomena of animal magnetism, hypnotism and ecstasy, frequently become developed under these circumstances.

Convulsive attacks are of frequent occurrence in hysterical women, and are often confounded with epileptic seizures, although there are numerous points of difference between the two.

The hysterical fit occurs almost always after painful emotions, maltreatment of children by their parents, or of wives by their husbands, terror on seeing some disgusting object or witnessing a convulsive attack in somebody else, or a sudden suppression of the menstrual flow; while epileptic attacks generally come on without any appreciable cause. The starting-point of the hysterical fit is generally in the epigastrium; while the epileptic attack occurs either without any warning, or with an aura of a different kind. The epileptic patient falls down as if

struck by lightning, no matter where he may be; the hysterical patient has almost always time to find a suitable place a bed or sofa upon which to fall. The epileptic convulsion is a sort of tetanus which does not resemble physiological movements, and scarcely ever lasts more than five or ten minutes: while the hysterical fit mimics physiological movements, and lasts often half an hour or more. At the end of the epileptic attack the patient falls into a deep coma, or he recovers consciousness at once, and feels shaken and exhausted; at the end of the hysterical fit, there is generally crying and sobbing. Urine of a peculiar character is passed, after the hysterical, but not after an epileptic, attack. The chief peculiarity of this urine is its great abundance, as it may amount to several pints at a time. It is clear and devoid of colour, almost inodorous and tasteless; it has a specific gravity of little over 1,000, and consists of scarcely anything but urinary water. The cause of this phenomenon is a spasm of the capillary vessels of the skin, which contain less blood than usual, and therefore throw additional work on the kidneys.

Acute hysteria is far more rare than the chronic form of the disorder, and is only developed if a powerful cause acts upon a system already predisposed to the disease; for instance, a great fright during menstruation. It is generally ushered in by one or several convulsive seizures, which are followed by delirium, paralysis, coma, and febrile symptoms. Where the patients recover their consciousness they complain of violent headache, thirst, loss of appetite, and a feeling of great lassitude; the pulse beats at the rate of above 100, and the temperature rises to 104° or even 106°. Diagnosis is sometimes difficult in these cases, which are not unfrequently confounded with typhoid fever or meningitis. Yet the result is only rarely fatal. In 1874 there died only 17 women of hysteria in England and Wales. The causes of death are generally asphyxia or syncope, from repeated convulsive fits, in which the patient does not recover from the coma.

In other cases death takes place by suicide or self-mutilation. Swallowed pins have caused fatal peritonitis. Generally, however, the attempts at suicide are not seriously meant; when the hysterical woman cuts her throat, jumps into the water, or swallows laudanum, she almost invariably does so in the pre-

sence of other people, of whom she is certain that they will do everything in their power to save the ebbing powers of life in the interesting patient.

The progress of hysteria is powerfully influenced by the events of life. If these be happy, the disease may come to a speedy end, but if the reverse, it may continue unabated to an advanced period of existence. The duration of hysteria is, therefore, very variable; and it is altogether much longer than is commonly believed. The symptoms of it may be very readily relieved, but the actual cure of the complaint is most difficult, as it depends to a great extent upon circumstances over which the physician has no control. Marriage has often been recommended as a cure for hysteria, but this can only act beneficially in a very limited number of cases. Pregnancy and lactation frequently aggravate the symptoms, and the illness and loss of children, the annoyances of housekeeping, and the bad temper of a husband, are apt to do the same. Moreover, there is great tendency to miscarriage in hysterical women; their children are often still-born, and, if they survive, they are generally delicate and sickly, and liable to inherit hysteria and other nervous affections. Marriage acts beneficially only where the circumstances of the patient at home are highly unfavourable, and where in consequence of marriage she is rid of all anxiety, embarrassments, and painful emotions.

Many patients only get well as age advances and sensibility becomes blunted; while others do not recover at all, or are troubled throughout life by the consequences of the malady. During the best years of their existence, they are subject to pain or convulsions, loss of voice or paralysis, they are unable to fulfil their duties, and a burden to themselves and others.

Strong moral emotions, affecting the will and the imagination of the patients, may cure hysteria temporarily or permanently, especially if they be of a sublime and exalting character. Such miraculous cures, apparently wrought by the agency of Faith, have been denied by some physicians, but are nevertheless as real as any cures obtained by other more tangible remedial agents. All is not humbug in the pilgrimage to Lourdes. Sir Benjamin Brodie has related the case of a patient affected with severe arthralgia, who had been in bed for several

years, but who, at the command of her spiritual adviser in the name of our Saviour, to get up and walk, actually did it. A striking instance of the same sort occurred in 1844 at Treves, in Germany, where a lady of rank, who had been completely paralysed for a number of years, was carried to the cathedral where the bishop had caused a sacred relic to be exhibited, at the sight of which she immediately regained the use of her limbs.

Partial or complete loss of muscular power is a frequent manifestation of hysteria, and invades with preference the left side of the body. Hysterical hemiplegia occurs either suddenly after painful emotions and hysterical attacks, or it creeps on gradually and unawares. It differs from hemiplegia from cerebral disease, by there being no distortion of the face, nor deviation of the tongue. The paralysis is scarcely ever complete, and mostly more severe in the leg than in the arm. It is subject to considerable and sudden variations under the influence of emotions or treatment. A woman affected with hysterical hemiplegia may, under the stimulus of great excitement, get out of bed, walk several miles, and perform other feats of power; and may then, after the excitement has subsided, relapse into complete immobility. No such thing is possible in a case of hemiplegia from intracranial disease. In the hysterical affection there is likewise generally hemianæsthesia, viz., great weakness or loss of sight and hearing, and of the sense of taste on the same side, and partial or complete anæsthesia of the skin; but no rigidity of muscles. Faradisation causes the muscles to contract, but the patients do not feel the passage of the current, unless this be one of great power. The electric contractility of the muscles is diminished only in cases of very long standing. Hysterical hemiplegia is often accompanied by retention of urine, sensations of pins and needles in the paralysed parts, febrile symptoms, with sleeplessness, and dyspepsia. The affection sometimes wanders about the body as in paralysis after acute diseases. At first there may be left hemiplegia; in a few months the left side will recover and the right side become affected; hemiplegia may then gradually become changed into paraplegia, and this into paralysis of the left hand.

Hysterical paraplegia is sometimes confounded with para-

plegia from myelitis and other diseases of the spinal cord or its membranes. It is, however, on its first appearance always accompanied by severe headache, showing the part played by the brain in the causation of the complaint, and many other symptoms which sufficiently distinguish it from paraplegia owing to structural disease.

By far the most common form of hysterical paralysis, however, is that which affects the vocal cord and the muscles of the larynx, and is known as hysterical aphonia. It almost always appears suddenly, after some violent emotion, or after taking cold; and it may last only an hour or two, or be protracted for years. The laryngoscope shows total absence of any structural lesion, the vocal cords being merely powerless, and incapable of movement.

In May, 1862, I treated an interesting case of this kind, together with Professor Czermak, who had just then introduced the laryngoscope into this country. It was the case of a domestic servant, aged 30, who had lost her voice two months before, on the sudden death of her master. The laryngeal mirror showed that both vocal cords were perfectly motionless, and that there was a large cleft between them. After two applications of faradism the patient could speak again, although still in a hoarse tone only. It was then discovered, by another examination with the laryngoscope, that the right vocal cord had to a great extent recovered, and approached the median line when the patient endeavoured to pronounce a prolonged 'ah,' but there was as yet no improvement in the left. By further treatment, the left vocal cord was also restored to its normal condition, and the patient entirely recovered.

Hysterical paralysis of the portio dura is rare, and is found together with anæsthesia of the skin and of the special senses of the same side, whereby it is easily distinguished from the ordinary form of facial palsy. The muscles of the pharynx and œsophagus may also lose their power, causing difficulty or impossibility of deglutition. The rectum is occasionally found paralysed and anæsthetic, which entails involuntary fæcal discharges. The power of the diaphragm may also be impaired. After bad hysterical attacks, the patient, at intervals, gasps for breath, and dyspnœa may be severe. The accessory muscles of

inspiration, more especially the scaleni, work hard; but the epigastrium and the base of the thorax do not project during inspiration, indicating a semi-paralytic condition of the diaphragm. The cardiac muscle may suffer in the same manner. Palpitations are frequent, and the pulse is sometimes hard and small, at other times full and soft, either from spasm or a semi-paralytic state of the vasomotor nerves. The skin is sometimes as cold as ice, and soon after becomes hot and perspires profusely. Pallor and blushing often alternate in the face. After convulsive attacks the pulse is sometimes imperceptible, and the heart's sounds may be inaudible. Occasionally death results from syncope, or trance from paresis of the heart.

The commencement and progress of all the different forms of hysterical paralysis leave no doubt on the mind that this is a functional disorder, and in no way owing to structural lesions in the nervous centres, the peripheral nerves, or the muscles. Affections which come on suddenly, which may vary in degree from day to day, and are sometimes cured by a single application of galvanism, cannot be due to organic lesions of important organs, but are produced under the influence of mental emotions, and chiefly determined by them in their further progress. In accordance with this view we find that in cases which have ended fatally, the most careful post-mortem examinations have failed to show any structural lesions to which the affection might have been fairly traced. In a few cases where anatomical alterations have been discovered, they were due to complications. Charcot has found sclerosis in the lateral columns of the cord in a woman who had for many years suffered from contractions of the limbs; but in cases of acute fatal hysteria, just as in fatal eclampsia, the microscope in the hands of most competent observers has shown all the different portions of the nervous system to be perfectly healthy. Whether the changes which undoubtedly take place in the nervous centres in this disease are chemical or simply molecular remains a problem for future investigations.

Paralysis is frequently accompanied with anæsthesia, but the latter may occur without the former. Of all the different kinds of loss of sensation which occurs in hysteria, *hemi-anæsthesia* is

the most interesting; and Charcot's researches on this condition are worthy of the greatest attention. It affects the head, neck, body, and limbs in a perfectly unilateral manner. All forms of sensation are in abeyance: common sensations, pain, touch, heat, cold, and electricity are unperceived; and temperature may be greatly reduced. The muscles, bones, mucous membranes, and nerves of special sense participate in the affection. Thus the sight is either diminished or absolutely lost; yet the ophthalmoscope does not reveal the slightest difference between the healthy and the blind eye. Where vision is not entirely suppressed it is less keen, and there is a concentric and general narrowing of the field of vision. This is particularly marked for the different colours; violet is lost first, then green, red, orange, yellow, and last of all blue. This crossed hysterical amblyopia is in all respects, except its causation, identical with cerebral hemianæsthesia from a lesion of the posterior part of the internal capsule, and the corona radiata.

The anæsthetic parts sometimes show diminished temperature and pallor; punctures made with large pins do not cause any blood to flow. The power of motion may be normal or even increased; there is no want of co-ordination, so that the finer movements, such as sewing, etc., are easily performed.

Charcot has drawn attention to a peculiar combination of hemi-anæthesia with *ovarian hyperæsthesia* and attacks of *hystero-epilepsy*, which forms one of the most singular manifestations of this proteiform disease.

There is spontaneous pain and tenderness in the ovary of the same side on which there is hemi-anæsthesia; and this is, as most other hysterical symptoms, on the left side. Attacks of hystero-epilepsy occur chiefly during menstruation, but also at other times, and are occasionally excessively frequent. They are ushered in by ovarian pain, palpitations, choking in the throat, tinnitus aurium, and throbbing in the temples, which is followed by coma and convulsions. The convulsions resemble the ordinary epileptic seizure, with a first period of rigidity, and a second of clonic convulsions, both being of shorter duration than the usual epileptic fit. A short stage of relaxation follows, but is in its turn succeeded by violent delirium, during which the patient throws herself wildly about, screams at the top of her voice,

laughs, cries, flies off the bed, and is most fearfully distorted. Finally there is a stage of exhaustion and general relaxation of the body, while the mind appears clear and bright. These attacks of hystero-epilepsy may be stopped at any stage by forcible pressure on the painful ovary. Tonic contraction of the anæsthetic limbs often occurs suddenly, and disappears as quickly. Metallo-therapy (i.e., the application of a chain of gold coins, or a bracelet of copper or iron, to the anæsthetic limbs), at first transfers the anæsthesia to the opposite side, and after a time removes it altogether.

Another symptom of frequent occurrence in hysterical women is neuralgia, which seems to select the joints, and more particularly the hip and knee. Pressure causes violent pain, but more so in the soft parts surrounding the joints than in the bones. Most cases of joint-affections which occur in the upper classes are of this description. They often last for years, and then get suddenly well, in consequence of a mental impression.

Hæmorrhage may occur from the skin and various internal organs, more especially where there is amænorrhœa, and where it forms the so-called vicarious menstruation; but it also sometimes takes place without any such relationship. If the bleeding proceed from the stomach, ulcer or cancer may be suspected; but the diagnosis is mostly rendered easy by the concomitant hysterical symptoms. Where this hæmorrhage occurs in the skin, it is called *stigmatisation*; and although this has been simulated by girls anxious to attract attention and excite interest, there can be no doubt that it sometimes occurs as a vasomotor neurosis under the influence of hysterical disturbance. In Louise Lateau the stigmata appear chiefly on the hands and feet, the forehead and the chest, and this is believed by the vulgar to be a repetition of the wounds of our Saviour on the cross. Bloody tears and perspiration may be seen under similar circumstances.

3. *Catalepsy, morbus attonitus.*

This condition is closely allied to epilepsy and hysteria, but presents certain peculiarities of its own which seem to constitute it a separate disease. It is not unfrequently seen in hysterical

women, but is also sometimes a symptom of chronic cerebral disease, which ultimately leads to insanity, and more particularly to melancholia and dementia. Such is generally the case when it occurs in males. In children it is occasionally met with as a sequela of tubercular meningitis. In advanced age it is rare, for most cases occur shortly after the development of puberty, in consequence of violent emotions, and more particularly shock to the affections. It has also been known to occur after severe fits of ague; and is occasionally followed by attacks of religious mania, mysticism, somnambulism, and ecstasy.

The fit of catalepsy generally comes on suddenly, and without a warning. The patient remains standing or sitting as if charmed; she has evidently lost her consciousness, and the life of relation has ceased for the time being. Circulation and respiration are barely perceptible, and all the muscles, but more especially those of the upper extremities, are in a state of rigidity. The muscles have a firm feel, and resist a change of position; but by employing more force the position of the limbs may be changed, and then remain unaltered for hours or even days. This condition has been called *flexibilitas cærea*. It sometimes lasts only for a few minutes; a kind of tremor is then seen to run through the muscles, and the limbs gradually drop into their natural position. There is a degree of anæsthesia, which is, however, not absolute, even in severe cases. Thus pricking and pinching may not be perceived, but faradisation by a powerful current will elicit signs of pain, and rouse the patient from her dormant state. I have found that faradisation of the skin of the face by metallic conductors is the most powerful agent for restoring consciousness in these cases.

The pulse if perceptible is slow and feeble, the temperature normal or diminished, and respiration retarded. Occasionally there are all the signs of suspended animation; and it is not impossible that patients while in this condition may have been buried alive. Faradisation is under these circumstances of great diagnostic value, because muscular contractions are by its means as readily produced as during health.

The fit may last from a few minutes to a few days, and show occasional remissions and exacerbations. Recovery is generally

gradual, but sometimes sudden, and the patient then behaves as if nothing unusual had occurred.

Uncomplicated catalepsy does not prove fatal. In cases where necropsies have been made, there has been as a rule complication with mania, epilepsy, chorea, tetanus, and other conditions, so that we are at present in the dark about any pathological changes which may occur in the nervous centres. It is, however, difficult to believe that any such alterations can be of a coarse nature, seeing the rapidity with which the cataleptic state originates and vanishes.

CHAPTER VII.

INSANITY.

I do not purpose in this chapter to enter into the pathology of insanity, which on account of its great importance is most properly looked upon as a specialty belonging to the alienist physician. I will, therefore, only for the sake of completeness, give the results of my researches on the prevalence of, and the mortality from, mental affections.

The following table shows the number of deaths from insanity which have been registered during six periods of five years each :—

Periods of five years	Deaths from Insanity	Percentage of Nervous Diseases	Percentage of all Diseases
1838–42	1,773	·84	·10
1843–46	—	—	—
1847–51	2,600	1·08	·12
1852–56	2,412	1·07	·12
1857–61	2,046	·75	·09
1862–66	2,959	1·01	·12
1867–71	3,861	1·25	·15

From this table it appears that the first lustrum showed the comparatively low mortality of 0·84; in the second a not inconsiderable rise took place, which was succeeded by a slight fall in the third, and a decided fall in the fourth, in which it reached a minimum of 0·75. A second rise then took place, and continued up to the last lustrum, which attained the maximum of 1·25. I have also found that each year, singly, of the last lustrum shows a rise over its predecessor, the maximum of 1·54 having been attained in the year 1871. The popular

notion that the mortality from insanity has increased during the last ten years is therefore shown to be correct.

The influence of sex on the mortality from insanity is shown in the following table:—

Periods of five years	Males	Percentage	Females	Percentage
1847–51	1,223	·51	1,377	·57
1852–56	1,087	·42	1,325	·51
1857–61	1,055	·38	1,394	·55
1862–66	1,391	·47	1,569	·55
1867–71	1,818	·59	2,043	·66

Women are therefore seen to die more from insanity than men, the mean average for them being 0·56 against 0·47 for men, showing an excess of 0·09 for women. The smallest excess was in the first lustrum, viz. 0·06, and the largest in the third, viz. 0·17. In men insanity showed a decided fall during the second and third lustra, while in the fourth and fifth an even more decided rise took place, so that the last lustrum is 0·08 higher than the first. For women the variations have been less striking. There was a slight fall in the second lustrum, a slight rise in the third, the fourth proved to be identical with its predecessor, and the last showed a somewhat more considerable increase.

The influence of age on the mortality from insanity is shown in diagram H. This disease is shown to carry off isolated victims as early as two years of age. The curve rises gradually in the subsequent periods, and more especially so at thirty-five, fifty, and sixty-five. At the latter age the curve attains its summit, and from thence falls rapidly to the end.

The influence of the sexes in this respect is shown in diagram I.

. The thick curve for men shows a slight ascendancy over the thin one for women until ten, after which the latter rises and keeps steadily overlapping the former, although otherwise the excursions of the two curves appear very analogous. The summit of the thick curve is at fifty, and that of the thin one at sixty-five.

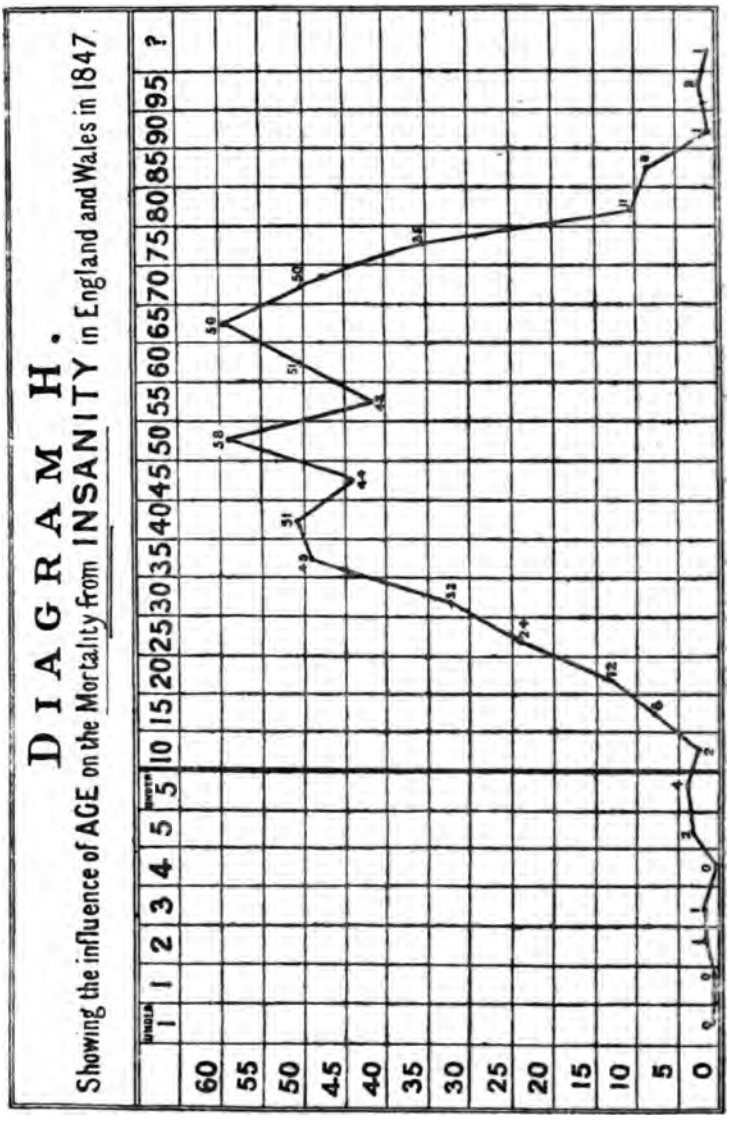


DIAGRAM H.
Showing the influence of AGE on the Mortality from INSANITY in England and Wales in 1847.
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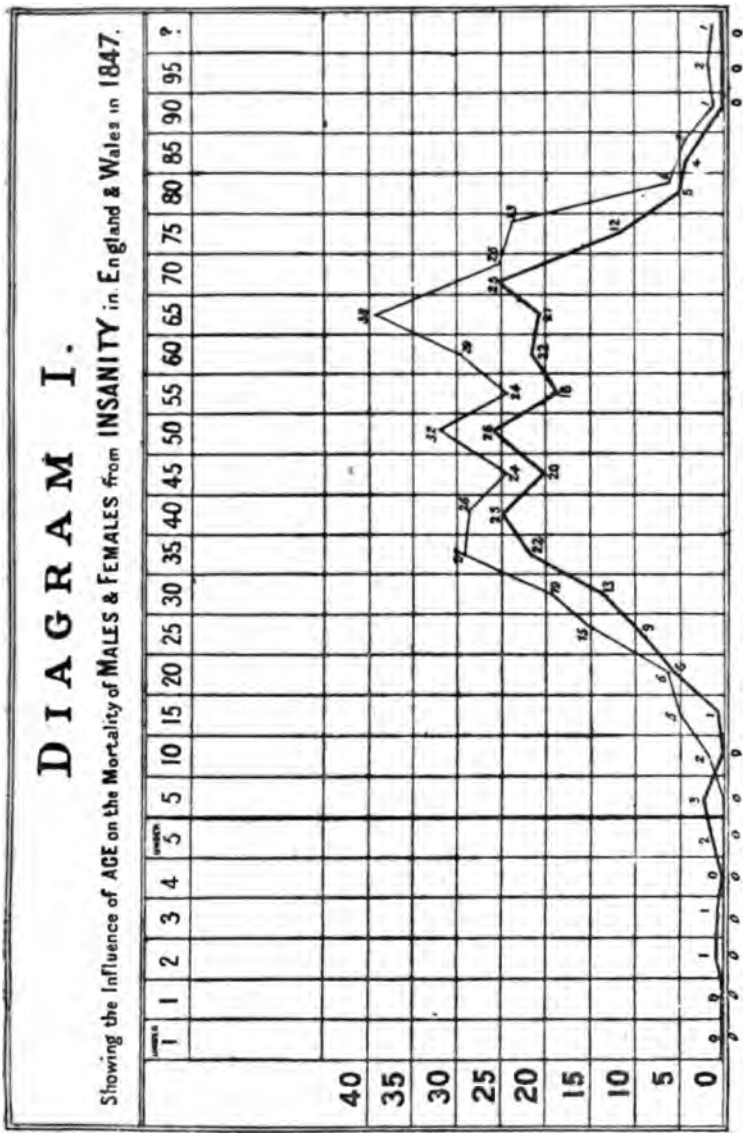


The thin curve shows the deaths of females, and the thick curve those of males.

Insanity is the only disease of which we actually know the number of living sufferers year by year; while for finding out the prevalence of other nervous complaints, as distinguished from mortality, we have to lean on clinical experience. It appears from the Reports of the Commissioners in Lunacy to the Lord Chancellor, that there were in 1859 under treatment in the various public and private asylums of England 36,762 lunatics; in 1869 this number had risen to 53,177; and in 1875 to 63,793. The deaths of all lunatics in 1859 amounted to 2,332, or 7·22 of the total number under treatment; in 1869 to 3,805, or 7·94 of the entire number; and in 1875 to 4,210, or 7·79 of the entire number.

It will be seen that the deaths of all lunatics, as given by the Commissioners in Lunacy, do not at all correspond to the deaths from insanity recorded by the Registrar-General; which is explained by the circumstance that in the former reports the deaths from all causes occurring amongst the lunatic population, and in the latter only the deaths from the various forms of brain-disease producing insanity, are included. Thus, the Registrar-General gives only 446 deaths from insanity in 1859, against 2,332 of the Lunacy Commissioners; the numbers being 722 and 3,805 respectively for 1869. This gives a proportion for the two years of 1 to 5·2; from which it appears that out of five lunatics only one actually dies of lunacy, while the other four die from other diseases.

The ratio of lunatics to 10,000 of the population was 18·67 in 1859, and had risen to 26·64 in 1875. This would at first sight appear to constitute a very considerable increase of insanity during the last seventeen years; but it should be considered that this increase of insanity, under care, has occurred chiefly among the pauper class; for, while during the period just mentioned the insane paupers have increased from 16·14 to 23·55 for 10,000 of the whole population, the private patients have during the same time increased only from 2·53 to 3·09. Nevertheless, there can be no question that there has been an increase, not only in the mortality from, but also in the prevalence of, insanity within the last few lustra.

The excess of females over males, which I have already adverted to in the *mortality* from insanity, is likewise per-

ceptible in the *prevalence* of that disease during life. The ratio for living male lunatics was 17·44 in 1859, against 19·85 for living female lunatics. In 1869 the respective numbers were 22·51 against 25·27; and in 1875, 24·87 against 28·32. These numbers show that the greater liability of women to go out of their mind has decidedly increased of late, as the excess over males was only 2·41 in 1859, and had gradually risen to 3·45 in 1875.

The average proportion of deaths from insanity to the living lunatic population, was, for the ten years from 1862 to 1871, 1 in 88.

CHAPTER VIII.

DELIRIUM TREMENS.

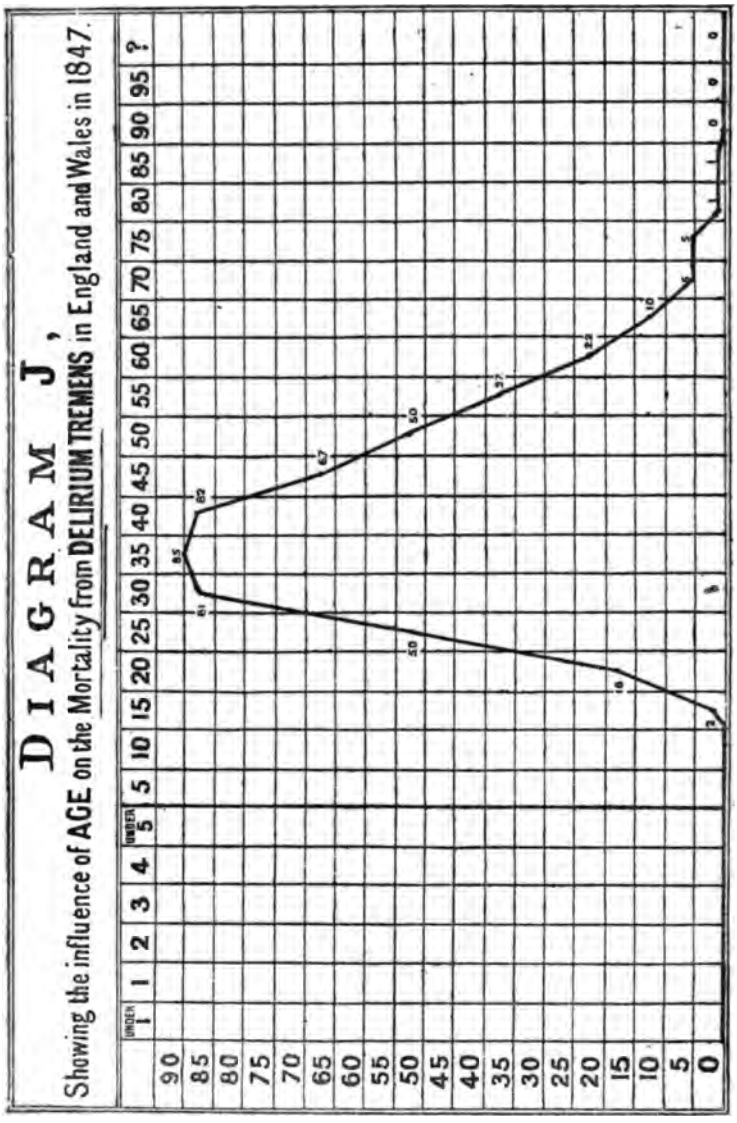
THE following table shows the number of deaths from delirium tremens which have been registered during six periods of five years each, and the percentage of mortality, first of nervous diseases, and second of all diseases :—

Periods of five years	Deaths from delirium tremens	Percentage of Nervous Diseases	Percentage of all diseases
1838–42	1152	·54	·06
1843–46	—	—	—
1847–51	2588	1·07	·12
1852–56	2534	·99	·12
1857–61	2325	·86	·10
1862–66	2633	·90	·10
1867–71	1948	·63	·07

From this table it is seen that the minimum was registered in the first period, and the maximum in the second. Since then there has been a fall which was particularly marked in the last lustrum.

The influence of age is shown in diagram J. There is a blank until after fifteen years; at twenty the curve begins to rise, advances more at twenty-five, and reaches its maximum at thirty-five. The greatest number die between thirty and forty. A fall then takes place, which becomes rapid after fifty-five, when it may be assumed that most of the unfortunate votaries of drink have either learnt wisdom from experience or died off. The minimum is reached at eighty-five.

This rate tallies on the whole with that found in the General



The top line of numbers denotes the periods of age at which the deaths have taken place.
The side line of numbers indicates the numbers of deaths at the different periods of life.

Hospital in Calcutta, from 1848–52, and the Medical College Hospital from 1851–53, as quoted by Dr. Aitken:—[1]

Ages from	Cases	Deaths
20–30	100	20
30–40	124	18
40–50	85	10
50–60	7	0
60–65	5	1

The influence of sex on the occurrence of delirium tremens may be expected to be, in the nature of things, a considerable one. Some observers have denied that this disease occurs in women; and in some continental countries, such as Italy, it is certainly never seen in them. In Norway and Sweden it is believed to occur in one woman to 170 men; and in Germany and France it is only exceptionally seen in women, by practitioners of the largest experience. In England and Wales, however, the rate of female mortality from delirium tremens is excessively high,[2] *the proportion being of one woman to eight men.* The numbers are as follows:—

Influence of sex on Delirium tremens.

Periods of five years	Males	Percentage	Females	Percentage
1847–51	2290	·96	298	·12
1852–56	2217	·86	317	·12
1857–61	2041	·75	284	·10
1862–66	2350	·80	203	·09
1867–71	1718	·55	230	·08

It is seen that for men there was a steady diminution during the first three lustra, a recrudescence in the fourth, and a further great fall in the fifth; and it is found that the mortality

[1] 'The Science and Practice of Medicine.'—London, 1870. Vol. i., p. 779.

[2] I have been informed that several hundreds of empty brandy-flasks are every day left behind in 'Ladies'-rooms' at the London railway stations by female passengers as they arrive, and that the sale of these flasks is looked upon by the attendants as their chief emolument.

from this disease has diminished by 0·41 as between the first and last lustra. For women the first two lustra showed an identical percentage, viz., 0·12, after which there was a steady fall, the last number being 0·08.

Dr. Aitken gives the rate of mortality in the Indian hospitals just mentioned as twenty-five men to one woman; which must also be considered very high. Seeing the great importance of the question of intemperance, I have taken the trouble to ascertain the death-rate from this disease in all the different counties of England and Wales, during the twenty-five years from 1847–72, and have arrived at very interesting results, which are shown in Diagram K.

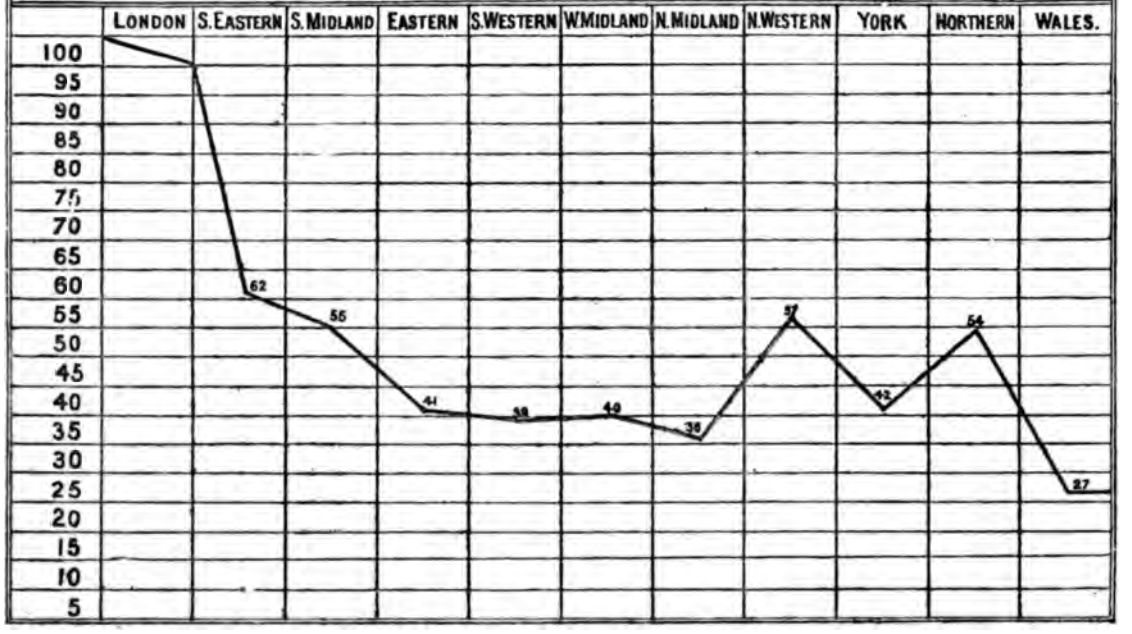
In doing this I proceeded in the following manner:—

The mortality from delirium tremens, and the entire mortality from all causes, were first extracted for each of the twenty-five years, for London and all the different divisions of England. The deaths from delirium were then divided into all deaths, when the death-rate for each year and each division appeared, and the results were then added together for the entire period. London, which was seen to be the highest of all, was then taken as a standard of 100, and the proportion of the other divisions to this found by the usual calculation. It is seen from the diagram that *London heads the list with* 100, the South-Eastern counties follow with 62, the North-Western with 57, the South Midland with 55, the Northern with 54, Yorkshire with 42, the Eastern division with 41, the West Midland with 40, the South-Western with 39, the North Midland with 36, and *last of all comes Wales with only* 27.

As the number of deaths from delirium tremens is known to correspond closely with the amount of strong alcoholic drinks consumed altogether by a population, it appears very significant that London, where we have seen nervous diseases to be at a comparatively low ebb, should consume proportionately so much more alcohol than Wales, where these maladies are so singularly rife. The question, therefore, naturally presents itself, whether the consumption of strong alcoholic drinks is really *always* prejudicial for the nervous system, as has been perhaps too sweepingly asserted by many well-intentioned men of late years; whether whisky is really 'the devil in disguise'; and

DIAGRAM K

Showing the comparative Mortality from **DELIRIUM TREMENS** in the Several Divisions of England & Wales, FROM 1847 TO 1871 RESPECTIVELY.



whether alcohol does not, in many instances, of course, only when taken in moderation, *act as a preservative against the invasion of diseases of the nervous system.* There are as yet not a sufficient number of accurate data at our disposal, to enable us to decide this point; but the facts I have brought forward are most suggestive, and will, I hope, lead other observers to investigate this question.

The pathology of delirium tremens is as yet obscure. There are, however, two points in it which were formerly believed to be facts, and have recently been shown to be fallacies, viz., the frequency of its occurrence in habitual drunkards, and its being the result of temporary abstinence from drinking, whereby the system lost its accustomed stimulus. Now it is certain that in the vast majority of habitual topers delirium tremens does not occur at all, unless a very prolonged debauch is committed; and it is equally certain that abstinence from drink will not induce the disease. Sudden abstinence may induce collapse, unless the patient be properly supplied with nourishing and easily digestible food, and, if necessary, with opium and hydrate of chloral; but it never leads to delirium tremens. On the other hand, patients sometimes evince great disgust to drinking at the commencement of the complaint, and this has most probably given rise to the erroneous idea of its being the consequence of abstinence.

The post-mortem appearances are those which have been described in the chapter on apoplexy from acute intoxication by alcohol (p. 134), to which in chronic cases are often superadded sclerosis of the cerebro-spinal centres, having caused imbecility, insanity, and paralysis during life; chronic catarrh of the bronchial tubes, which is sometimes combined with emphysema, and has during life given rise to asthma and other respiratory troubles; enlargement of the liver, granular degeneration and cirrhosis of the organ; atheromatous disease of the heart and arteries, and lastly the atrophic form of Bright's disease of the kidneys. The blood has been found charged with fat, so that the serum appears milky-white, and loses this colour when treated with ether, showing that it is not owing to white blood-globules or particles of albumen. This state of the blood explains the well-known fact that most habitual topers get very

fat, and that ill-nourished, wasted children rapidly put on flesh when alcohol is given them.

Symptoms of chronic alcoholism generally precede the outbreak of delirium tremens for a considerable time; and amongst these the most noteworthy are want of appetite for solid food, mental decay, and sleeplessness. The first signs of commencing delirium are an increase in these symptoms, so that there is loathing to food, and often to drink, loss of control over the mental faculties, and complete insomnia. The restlessness both of body and mind soon reaches an extreme degree; the hands shake, the tongue is tremulous when protruded, and the patient is altogether in a state of the greatest agitation and alarm. Hallucinations of vision now begin to terrify him: he sees rats and mice swarming about the room ready to devour him, snakes wriggling up the bedclothes, and cockroaches passing in processions; and he is constantly busy pushing away his imaginary enemies. Hallucinations of hearing are less frequent. The bowels are confined; there is pain and tightness about the region of the liver; the urine is scanty and loaded with lithates, and sometimes contains blood and albumen. The skin is cold and clammy, the pulse small and compressible at the wrist, but full and throbbing in the carotid arteries. The tremor of the hands and upper extremities generally, from which the disease has received its name, is in many cases incessant, and sometimes prevents the pulse from being counted at the wrist; but in some instances tremor is quite absent, more especially where the affection occurs in young and otherwise robust persons, in whom agitation, hallucinations and sleeplessness, form the most prominent symptoms. After the patient has been in this condition for three or four days, a state of exhaustion supervenes, which either merges into collapse and death, or into a tranquil sleep, from which he awakens much refreshed and easier in his mind, although in a state of great feebleness both physically and mentally. Reformed habits sometimes date from such an outbreak of illness, but more frequently the patient after a time returns to the bottle, and ultimately falls a victim, either to a fresh attack of delirium, or to apoplexy, general paralysis, and liver and kidney disease.

CHAPTER IX.

TETANUS.

Concerning tetanus the information given by the Registrar-General is not so complete as with regard to other diseases. Tetanus was at first registered together with other affections of the nervous system, but was in 1858 expunged from that class, and 'idiopathic tetanus' only was then registered in the supplementary tables. From 1863 'traumatic tetanus' was added to the idiopathic form of the disease.

Mortality from Tetanus.

Periods of five years	Deaths from Tetanus	Percentage of Nervous Diseases	Percentage of all diseases
1838–42	529	·25	·030
1843–46	—	—	—
1847–51	643	·26	·031
1852–56	714	·27	·034

Mortality from

Periods of five years	Idiopathic Tetanus	Traumatic Tetanus	Total
1858–62	225	—	—
1863–67	146	549	695
1868–72	123	613	736

The influence of age on the mortality from tetanus is shown in Diagram L.

The curve shows that affection to be comparatively frequent

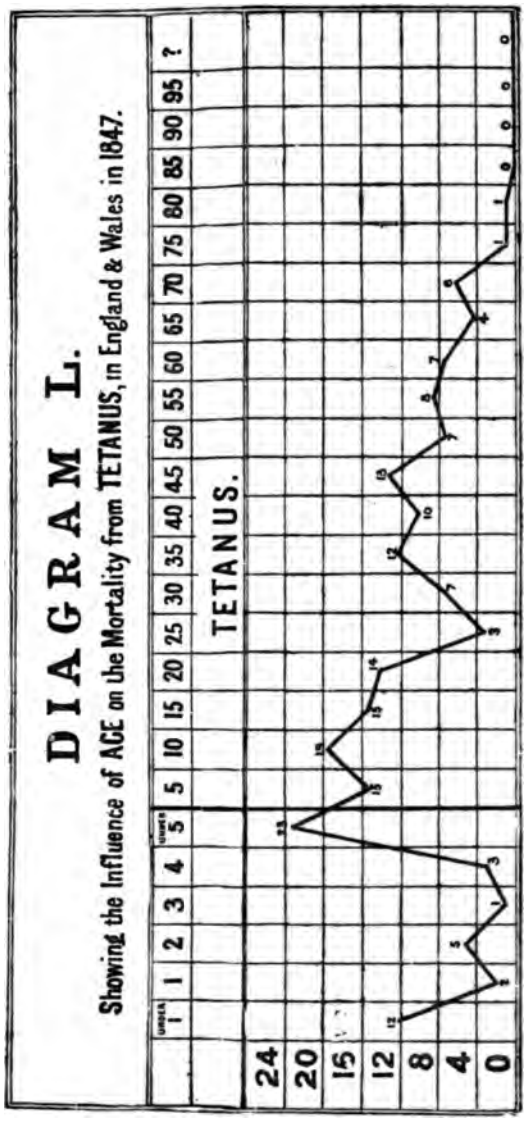


DIAGRAM L.
Showing the Influence of AGE on the Mortality from TETANUS, in England & Wales in 1847.
TETANUS.
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be separated, but only by an effort, and the tongue may still be protruded between the teeth. As time goes on, however, the symptoms increase in intensity; the neck becomes stiffer, the jaws are so perfectly 'locked' that the upper and lower teeth are pressed against each other, and that no solid food and only little liquid can pass through them. The spasm in the masseter and temporal muscles is so severe that neither the patient's efforts nor any external force can open the mouth. The rigidity of the tongue is such as to interfere considerably with articulation, so that the voice of the patient is quite altered. The portio dura likewise becomes implicated; there is risus sardonicus; the eyebrows are drawn up; the eyes are staring and immovable; and there is generally an expression of terror in the physiognomy. The face is pale, and the muscular contractions give the patient an aged look.

The rigidity now spreads to the body, and more particularly to the long extensors of the spine. The head is drawn backwards, and becomes ultimately fixed in this position, so that the patient is unable to move it to the side. The spine is curved forwards, so that the body rests on the back part of the head and the sacrum; and the chest is also arched forwards, whereby its broadness appears increased. After a time the abdomen likewise becomes affected; the epigastrium is drawn in, and the belly is hard and flattened. The rigidity of the abdominal muscles interferes considerably with respiration, inasmuch as it prevents the descent of the diaphragm, and admission of air into the lungs; while expiration is rendered difficult by rigidity of the thoracic muscles. The patient is therefore unable to cough, and mucus is apt to accumulate in the bronchial tubes. As the glottis is half closed, and neither the inspiratory nor expiratory muscles can act properly, dyspnœa is the result, which often reaches an extreme degree, so that the patient dies by asphyxia.

The upper extremities frequently escape, while the lower limbs are generally in a state of rigidity, which chiefly affects the extensors. There may be spasmodic erection. The muscles ultimately become as rigid as iron, and voluntary as well as passive movements are impossible. The expulsive action of the bladder is generally impeded, partly by spasm of the sphincter,

and partly by rigidity of the abdominal muscles. The retention of urine, which is thus brought about, ceases, however, in a warm bath. The bowels are habitually confined, no doubt likewise from rigidity of the abdominal muscles, and from spasm of the sphincter ani. When fæces are brought away by enemata, they are found to be most offensive.

Where sleep occurs, and also when the patient is put under the influence of chloroform, or some other anæsthetic, the muscular rigidity ceases; but it recommences at once when consciousness is restored.

In some instances the continuous tetanic rigidity of the muscles constitutes the principal symptom; while in another class of cases, convulsive fits come on, to add to the distress of the patient. These paroxysms closely resemble the convulsions observed in strychnia-poisoning, and occur much in the same manner; i.e., they may be, as it were, spontaneous discharges of the unduly excitable nervous force, or they come on reflexly from causes which act as irritants on the sentient nerves. A mere touch, as when the doctor feels the pulse; shaking of the floor when the door is opened or shut; or intended movements, such as swallowing, may cause such fits to break out. This is evidently owing to convulsibility of the medullary centre, for the movements are co-ordinated, and the heart's action is generally interfered with. The inhibitory centres of reflex action in the corpora quadrigemina, however, do not under these circumstances invariably lose their controlling influence altogether; for if the patient's attention is drawn to a sentient impression just previous to its occurrence, he is often able to suppress the attack.

The first stage of the convulsive fit is merely an exaggeration of the rigidity which habitually exists in tetanus. The opisthotonus becomes more marked, the head is pressed further back into the pillow, the tongue is pressed between the teeth and bitten. Respiration is rendered even more difficult from constriction of the pharynx and glottis, and greater spasmodic contraction of the thoracic and abdominal muscles, whereby the descent of the diaphragm is more impeded; the countenance is more anxious and terrified, and the complexion livid. Foam appears at the mouth, and the urine is often projected to a

considerable distance. The pain of these convulsive seizures is fearful, and the patient dreads their occurrence more than anything else. They last generally a few minutes, and occur every half-hour or hour. They become more frequent where a fatal issue is about to occur, while they gradually decrease in number when recovery is to be the patient's lot. The fit generally ceases with chronic convulsions and tremor.

The intellect is not affected, but in exceptional cases delirium supervenes early, which must be looked upon as highly unfavourable. As a rule, there is sleeplessness and restlessness, with pain in the epigastrium. The patient cannot find a comfortable position, and the interference with respiration causes a feeling of suffocation and anxiety. At the same time there is much thirst and hunger, which cannot be stilled from difficulty of swallowing. The thirst is particularly distressing; the tongue is dry and coated by a thick fur; the saliva is scanty and viscid; but digestion does not appear to be impaired.

The heart's action is at first not altered, for the pulse is in the beginning regular, and beats at the usual rate; but it gradually rises to 90, 120, and towards the end becomes too quick to be counted. It is also accelerated by ten or twelve beats during the convulsive paroxysm, and becomes slower after the fit is over. The quick pulse of the latter period of the disease is no doubt owing to irritation of the sympathetic fibres coursing in the spinal cord. The bloodvessels are in consequence of this so contracted that even during the amputation of a limb, which has sometimes been resorted to for the cure of tetanus, occasionally not a drop of blood has been lost. During a fit the heart may stand still during diastole, from irritation of the root of the pneumogastric nerve in the medulla, and the patient may thus die in a fit.

Respiration is generally not accelerated, but difficult and forcible, and may, as we have seen, become arrested during a convulsive attack. Tracheotomy has been resorted to under these circumstances, but the results have been unfavourable, as the patient is unable to expectorate the mucus which collects in the air-passages, and is then carried off by bronchitis.

The temperature remains normal as long as there are no convulsive seizures, unless there should be complications con-

nected with the original injury or surgical fever. In the fit there is at first a fall of heat, but afterwards a rise, which continues for a time after the attack is over. Each fresh fit sends the mercury higher up, and towards the end the temperature may reach 110° and even 112°. It is a singular fact that before a convulsive attack the temperature of the muscles should be lower than that of the rectum, while after the fit is over it should be higher in the muscles. This would lead us to think that the heat is developed in the muscular tissue, in consequence of excessive contraction of the same; but it is probable that the heat-regulating centre in the spinal cord (p. 33) is likewise of influence in this respect. In some cases the heat continues to rise for some time after death, which some physiologists are inclined to ascribe to the rapid post-mortem coagulation of myosine in the substance of the muscles; but may also be owing to the diffusion of heat from the muscles to the skin and rectum.

The skin is generally covered with profuse perspiration, more especially after fits. As the arteries are contracted, the pressure of blood is increased in the cutaneous vessels, leading to increased action of the sudoriferous glands; and sudamina are sometimes developed. The urine, on the other hand, is generally scanty, and as the patient can drink but little, and such liquid is lost by the skin, the density of the urine is very high, and it deposits a large quantity of urates on standing. There is no excess of urea; but albumen and sugar have been found in a few cases.

Hippocrates maintained that if the patient lived beyond the fourth day, the case was sure to end in recovery; but such is not the case, for many patients die between the fourth and tenth day of the disease, and a few of them even weeks after its outbreak, from exhaustion or certain complications. In Friedrich's 128 fatal cases 83 patients died within the first four days, and the other 45 at variable intervals after the fourth day. Frequent convulsive seizures are incompatible with life, and death takes place either during a fit, from syncope or asphyxia, or the attacks cease suddenly, delirium supervenes, there is complete prostration of force, the temperature and pulse rise rapidly, and the patient dies comatose. Rigor mortis commences

sometimes almost immediately after death, and the jaw does not become unlocked until cadaveric rigidity has disappeared. But if not immediate, rigor is always rapid in its occurrence, lasts longer than usual, and is very strongly marked, probably in consequence of the excess of lactic and other acids which are developed by the hyperactive condition of the muscles, and the coagulation of myosine.

In private practice the results are more favourable than those obtained by military surgeons, owing to the better circumstances of private patients. Surgeon McGrigor saw in the Peninsular war only a few recoveries out of several hundred cases; and Demme, in the war between Austria and France in 1859, had 80 deaths out of 86 cases.

The longer the disease lasts the better are the patient's prospects; but even when recovery has fairly set in, it is always gradual and protracted over weeks and even months. Certain forms of paralysis are apt to remain after recovery, viz. paralysis of the portio dura, which is most likely owing to ascending neuritis; and paralysis of certain muscles of the extremities, which, no doubt, arise from the persistence of similar lesions in the cord, as Dr. Lockhart Clarke has found in fatal cases of tetanus.

CHAPTER X.

CHOREA.

CHOREA, the dance, or St. Vitus's Dance, appeared in the middle ages as an epidemic disorder; and it received the name of St. Vitus's Dance from the Sicilian saint St. Vitus, whose mortal remains were believed to rest in the convent of Korvey, in Westphalia, to which pilgrims from all parts were attracted in order to be cured of the dancing mania. Synonyms of chorea are ballismus, orchestromania, chorea Anglorum, epilepsia saltatoria, muscular insanity, folie musculaire, danse de St. Guy, Veitstanz.

The deaths from chorea in England and Wales during six lustra were as follows:—

Periods of five years	Deaths from Chorea	Percentage of Nervous Diseases	Percentage of all Diseases
1838–42	150	·07	·008
1843–46	vacat	—	—
1847–51	248	·10	0·012
1852–56	316	·12	0·015
1857–61	289	·10	0·013
1862–66	339	·11	0·014
1867–71	373	·12	0·013
Total of thirty years	1,715	·10	·012

It will be seen from this table that the minimum mortality of chorea was registered in the first lustrum. The fatality of the disease rose in the second and third lustra, fell in the fourth, and rose again in the fifth and sixth. It is, however, seen that the percentages of mortality of nervous and of all diseases do not completely tally.

The prevalence of chorea during life as compared to its fatality, appears to vary in different countries. In English practice it is found that there is on the average one death to a

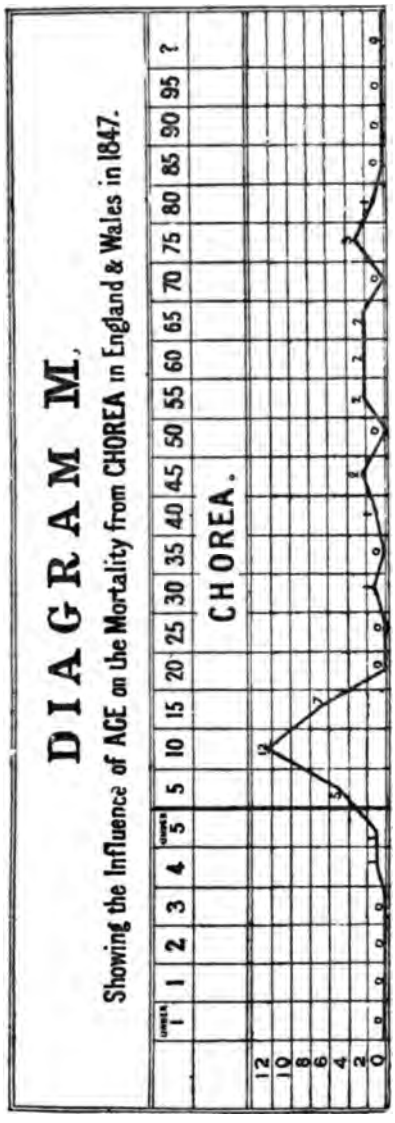
DIAGRAM M.
Showing the Influence of AGE on the Mortality from CHOREA in England & Wales in 1847.
CHOREA.
Under 1
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Under 5
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10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
95
?
12
10
8
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hundred cases, while Sée found in Paris a mortality of more than five per cent. in children; and Wenzel in Germany a mortality of 27 per cent. for pregnant women.

That *age* has a great influence on the production of chorea has been noted by all previous observers. It may be said that two-thirds of the cases occur between the second dentition and the development of puberty; but it also occurs in newly-born infants and late in life. Some time ago I had a case of severe bilateral chorea under my care at the hospital, in which the affection had come on immediately after birth. The patient was a boy aged 14, a pauper orphan, about whom no history could be obtained. He had spent his life in the workhouse, and had always been violently choreic; and it may be imagined that his mother must have gone through severe trials previous to the birth of her unfortunate child.

The table inserted at p. 294 shows that during the first three years of life no deaths from chorea took place, and that the disease was most fatal between five and fifteen years of age. Between 20 and 30 the mortality subsided altogether; between 30 and 34 there was only one death; between 35 and 39 none, and between 40 and 44 only one. These numbers show that chorea is not so very fatal to pregnant women as some observers have assumed.

The great influence of sex on the occurrence of this disease has been remarked by all previous observers. M. Sée found that of 531 cases admitted into the hospital for sick children in Paris, 393 were females, and 138 males, that is, a proportion of 2·8 to 1. In England and Wales matters are very similar, as shown by the following table:—

Deaths from Chorea.

Periods of five years	Males	Percentage	Females	Percentage
1847–51	68	·02	180	·07
1852–56	102	·03	214	·08
1857–61	82	·03	207	·07
1862–66	93	·03	246	·07
1867–71	123	·03	250	·08
Mean Percentage of five lustra .	—	**·03**	—	**·07**

The chief symptoms of chorea are more or less violent, and nearly incessant contractions of voluntary muscles or sets of muscles, which occur either spontaneously or when voluntary movements are intended; they are devoid of purpose, improperly co-ordinated, cease only during sleep, or when the patients are under the influence of anæsthetics, and are not accompanied by loss of consciousness.

Chorea comes on either suddenly, or after certain premonitory symptoms which show a disturbance of the central nervous system. The temper of the patient undergoes a marked change: he becomes morose, irritable, fretful, lazy, forgetful, and complains of headache, dizziness and chilliness. Occasionally there are signs of hyperæsthesia or paræsthesia on the parts of the nerves of special sense and of common sensation; palpitation of the heart, loss of appetite, and general restlessness. After this condition has lasted a short time, the characteristic feature of chorea becomes developed, viz., a general restlessness of the whole muscular system of the body, the only muscles which escape being the sphincters of the bladder and rectum, and the small muscles of the external ear. In some cases the muscular dance is so severe that voluntary movements are utterly impossible, and the patient becomes helpless. He cannot stand, sit, or lie down with comfort, and has to be dressed and fed like a baby. If he attempts to stand, the knees give way and he drops to the ground; and the whole body is so plunged, jerked and shaken about that he cannot even sit still on a chair, and is thrown out of bed when lying down. The clothes are so much pulled and rubbed about the body that abrasions of the skin and eczematous eruptions are produced by the incessant irritation.

In most cases, however, voluntary motion is not entirely abolished, but only impeded. Although the involuntary motion is incessant, the patient nevertheless succeeds in carrying out voluntary movements. It is true that he performs them in an awkward, clumsy, and roundabout manner; the movement which is intended is commenced, but is, before it can be carried out, interrupted by spasms. The patient then commences to manœuvre, and succeeds after a time in accomplishing his purpose; but at other times the effort of the will appears to

intensify the spasm, which from having been limited to the face and hand may then involve the whole body. A simple effort on the part of the patient to subdue the spasm, and to keep his face and limbs quiet, is often sufficient to increase the violence of the choreic twitches.

The facial muscles are pre-eminently affected in this disease, for all kinds of physiognomical expressions are produced, according as the spasm affects the different muscles one after another, and the general impression produced on the observer is decidedly grotesque. The patient first frowns, then laughs, squints, winks with his eyes, stares, moves his nose from one side to another, grinds his teeth, puts out the tongue, and then as quickly draws it back again, bites the tongue, lips and inside of the cheeks, and grins like a monkey. In the muscles of the lower jaw the spasm is sometimes so violent that teeth are pulled, or rather kicked, out by it. Mastication therefore becomes difficult or impossible, and the first act of deglutition is impeded. The speech may become quite unintelligible. The patient begins a sentence, but cannot finish it, because the tongue is in his way; sometimes he is only able to pronounce one syllable at a time. He cannot sing because respiration is jerky and explosive, the air is not properly husbanded, and although great efforts may be made, there is no result except great fatigue, from excessive disorder in the co-ordination of the muscles. Ziemssen[1] has seen choreic movements of the abductors and other muscles of the vocal cords, and insufficient tension of the latter by the laryngeal mirror, and this explains why the voice should be changed in character, monotonous, and lowered in pitch.

One or both upper extremities show similar symptoms of muscular madness. The shoulder is drawn up and pushed down again, the arm jerked about in an incongruous fashion, and the hand and fingers are thrown hither and thither. The patient cannot take a cup of tea to his mouth without manœuvring in a desperate fashion, and is yet apt to spill the contents; he is unable to work, write, play on musical instruments, and button his sleeves. That the diaphragm and abdominal musles suffer is shown by unequal and irregular respiration, and the pelvis

[1] 'Handbuch der speciellen Pathologie und Therapie.'—1875, vol. xii., II., 2, p. 414.

and lower extremities are so unsteady that locomotion becomes difficult or impossible.

In mild cases all these contractions occur only when the patient is excited or intends to do something, while in severe cases they are constant and spontaneous, but even then increase in force by excitement or attempted voluntary motion.

An interesting form of this disease is that known as *hemi-chorea*, which is limited to one side of the body. It is most frequently seated in the left side, which is also, as a rule, more severely affected in bilateral chorea than the right. Of twenty-two cases of hemi-chorea which have been under my care at the hospital, there were sixteen of the left, and six of the right side.

Hemi-chorea is sometimes complicated with hemi-anæsthesia, and preceded or followed by hemiplegia. Unilateral facial paralysis of cerebral origin is then likewise present.

In some cases the nerves of the affected parts are tender to pressure, and there may be spontaneous pain in their sphere. Certain portions of the spine may also be unduly sensitive. The motor nerves and muscles are extremely excitable both to the galvanic and faradic current, just as in certain cases of hemiplegia owing to irritative cerebral lesions. A slight current will then cause tetanic rigidity of the muscles, which occasionally persists for some time after the application. While the physiological formula for the continuous current is contraction on making the direct current, it is found that in chorea a contraction appears not only on making, but also on breaking the circuit. Ordinary reflex excitability may likewise be increased.

There is generally muscular rest during sleep, but this is by no means constant. In many instances there is great difficulty in going to sleep, owing to continuance of the muscular spasm, and sleep when obtained is not profound, but broken. The patient moans and grunts, and throws himself about in a restless manner. On awakening in the morning there is at first muscular rest, but the spasm soon recommences, more especially on getting up and making an attempt to dress. The spasm ceases, however, completely when the patient is placed fully under the influence of chloroform or hydrate of chloral.

Mental symptoms are rarely absent, and become more

developed when the disease lasts a considerable time. The judgment is perverted, and ideation slow; there is indifference and want of respect to parents and other near relations; the memory is impaired, and there is great want of attention. Some patients are shy and timid, others quarrelsome and boisterous, but all are more or less silly. In some cases mania and imbecility follow after a time. These symptoms are not only observed towards the end, when they might be ascribed to exhaustion and anæmia, which are apt to come on then, but are often present at an early period of the complaint.

The pupils are generally large and sluggish in their movements. The pulse is sometimes normal, but it may be very quick, hard, small, and intermittent. The body-heat is, in spite of the incessant muscular motion, not increased. Appetite and digestion suffer especially in protracted cases, and the patient generally becomes emaciated and loses in weight. The bowels are confined, the urine is scanty, and contains an excess of urea. The skin is dry, and sometimes covered with an eczematous eruption.

The average duration of chorea is from two to three months, but this can be considerably shortened by appropriate treatment. Recovery is sometimes interrupted by unfavourable emotional influences, more especially fright or unkindness, and relapses are liable to occur even after long intervals, particularly during spring. There is rarely more than one relapse; but in some cases a tendency to irregular muscular contractions remains for a long time. Where chorea becomes established for life, and is associated with paralysis, epilepsy, melancholia and mania, it has to be looked upon as only one of the symptoms of extensive brain disease.

Death from St. Vitus's dance is rare, and occurs either from great intensity of the disease, or by complications, such as endocarditis, pericarditis, embolism of cerebral arteries, meningitis, encephalitis and myelitis. Where uncomplicated chorea ends fatally, the symptoms are severe from the commencement; there is complete sleeplessness, violent muscular commotions by night and day, which are suddenly followed by collapse, with a small and feeble pulse, involuntary evacuations of the urine and fæces, and coma. In some cases the choreic move-

ments disappear shortly before death, while in others they last, although less severely, up to the instant of dissolution.

The exciting causes of St. Vitus's dance are frequently such as act upon the emotions, viz., fright, fear, terror, and anxiety, and it occurs particularly in children who have inherited the nervous temperament from their parents. Hysteria of the mother is most effective in this respect. Chlorosis, anæmia, dysmenorrhœa, and scarlet fever often precede the outbreak of chorea; and it would appear to be sometimes owing to the pernicious habit of masturbation. It is no doubt occasionally contagious, but only to those children whose nervous system is excessively impressionable. In some cases the disturbance of the nervous system which causes the outbreak of chorea, is not of a mental, but of a reflex nature, and owing to some peripheral irritation spreading to the nervous centres. We therefore find it during pregnancy, where it comes on in consequence of the development of the gravid uterus, and ceases after delivery. It generally makes its appearance in the first half of pregnancy, especially in primiparæ, and between the ages of twenty and twenty-four. It is observed not only in those who have already suffered from it during childhood, but also in those who never had it before. It generally disappears after delivery, but may come on again in every subsequent pregnancy. Occasionally it is developed in the lying-in period; and is also connected, as in children, with rheumatism and endocarditis. It seems frequently to give rise to abortion. We also speak of chorea being reflexly induced where it comes on after injury to peripheral nerves, and cicatricial shrinking of the parts. In such cases it sometimes ceases after excision of the scars.

In a very large class of cases, however, rheumatism must be looked upon as the exciting cause of chorea. Most frequently it is rheumatic fever, but even simple rheumatism of the muscles without febrile disturbance and articular affection may give rise to it.

The pathology of chorea is as yet unsettled, although much progress towards the solution of this problem has been made in recent years. That the brain is the principal, if not the exclusive seat of the lesion producing chorea, was taught by the late Dr. Todd, who in support of his view pointed to the fre-

quently unilateral form of the disease, and its occasional complication with hemiplegia. A step further was made by Dr. Russell Reynolds, who adopted Dr. Todd's theory of the cerebral, not spinal, origin of chorea, and pointed to the corpora striata as the chief seat of the disease. Nevertheless, some recent French observers, such as Chauveau,[1] Carville, and others, have endeavoured to prove that the exclusive seat of chorea is in the posterior horns of the grey matter in the centre of the cord, and that the brain has nothing whatever to do with it.

The nature of the anatomical lesion has likewise been differently stated by various observers. Thus we have softening of the occipital convolutions of the left cerebral hemisphere, and sclerosis of the posterior columns of the spinal cord (Cruveilhier); proliferation of connective tissue in the cord (Rokitansky); capillary embolism of the basal ganglia and their surroundings (Broadbent); softening of the right and in a less degree of the left middle cerebral lobe without embolism (Tuckwell); embolic thrombosis of certain cerebral arteries and softening of the anterior and middle cerebral lobes and the dorsal portion of the spinal cord (Gray); cicatrices at the posterior end of the thalamus opticus and nucleus caudatus, and at the posterior portion of the corona radiata, as well as effusion of blood in the thalamus opticus, and in one of the anterior corpora quadrigemina (Charcot); diffuse changes affecting not only the thalamus opticus, corpus striatum, the insula and claustrum, but also granular proliferation in the connective tissue of the peripheral nerves and along the vessels of the spinal cord (Elischer); hyperæmia in the small vessels of the central ganglia, and occlusions of the same, these being partly fibrinous plugs and partly composed of rounded concretions, apparently of an albuminoid nature (Charlton Bastian); thrombosis of the vessels of the grey matter of the convolutions of the post-parietal and occipital lobes as well as perivascular spaces and degenerated nerve-cells (Stretch Dowse).

The embolic theory of the production of chorea has been supported chiefly by Dr. Parkes, Dr. H. Jackson and Dr. Stephen Mackenzie; but the circumstance that in very numerous cases

[1] 'Archives générales de médecine,' Mars, 1866. 'Comptes rendus,' etc., vol. 60, p. 1046. 1870.

of chorea such able pathologists as Drs. Wilks, Moxon, and Dickinson have been unable to find emboli, as well as the circumstance that in undoubted embolism of the cerebral arteries there is never any appearance of chorea, must militate very strongly against this hypothesis.

A most valuable contribution to the pathology of chorea has recently been made by Dr. Dickinson [1] who found in seven cases remarkably constant post-mortem appearances, viz., hyperæmia or distension of all the vessels of the brain and cord, but more particularly of the small arteries, which was succeeded by effusion of blood with consequent irritation and injury of the surrounding tissue. The changes in the two sides of the brain and cord were generally symmetrical. The parts of the brain which were most affected were those between the base and the floor of the lateral ventricles in the track of the Sylvian arteries; the substantia perforata, the corpora striata, and the beginning of the Sylvian fissures. In the cord the pathological changes were chiefly marked in the central portion of each lateral mass of grey matter, comprising the root of each posterior horn. In none of these instances was there any trace of embolism, viz., decolorised fibrine, detached clots, or signs of impaction. Moreover, the constancy with which these changes repeated themselves in certain positions, and the equality with which they affected both sides of the body, appeared to be conclusive objections to this hypothesis. The corpora striata, for example, were affected with almost absolute symmetry, although they receive their blood respectively from the right and left carotids, and different portions of the aortic arch. These changes seem to us to explain satisfactorily the symptoms observed during life in choreic patients.

In most fatal cases of chorea, mitral *endocarditis* is discovered, and this is sometimes associated with articular rheumatism, at other times not. There can be no doubt that some, especially French observers, have more frequently diagnosed endocarditis than there was real reason for it; for systolic murmurs may be present without endocarditis, and may be simply indicative of deficient innervation of the muscular substance of

[1] 'Medico-Chirurgical Transactions for 1876,' p. 1.

the heart without structural disease. Anæmia is sometimes found to exist in the commencement of St. Vitus's dance, and more especially where the disease has lasted a considerable time. Nevertheless organic mitral murmurs are most frequent in chorea, and are accompanied with intensification of the second sound of the pulmonary artery, and hypertrophy of the left ventricle. In some cases these murmurs disappear after a time, no doubt from the quantity of lymph deposited on the valve having been small, so that there could be complete absorption of it, and recovery of the valve.

The question therefore arises whether chorea causes endocarditis, or endocarditis chorea? The late Dr. Kirkes believed endocarditis to be the true cause of the muscular dance, the inflammatory products being conveyed from the valves to the blood, and then setting up irritation of the nervous system. But it is difficult to believe that in those cases where fright is the unquestionable origin of chorea in previously healthy children, endocarditis should have been present before, or that it should have come on subsequently without any relation to the chorea. Dr. Dickinson has rendered it probable that the irregularity of the heart's action which is often present in this disease—true chorea of the cardiac muscle—and the regurgitation of blood, which is the consequence of it, may cause the deposition upon the valve which passes for endocarditis. In such cases the fibrine is deposited in small soft beads on the inner surface of the mitral valve; and it appears to us fair to assume that chorea causes the valvular beading, and that endocarditis has no real influence in the causation of chorea.

Our views on the pathology of chorea may therefore be summed up as follows:—

1. *In a large class of cases chorea is owing to that alteration in the composition of the blood which is associated with rheumatic fever*, and which is known to affect the nervous centres as well as other organs.

2. *In another large class of cases chorea is produced by direct irritation of the nervous system*, which is either purely mental in its nature (fright, etc.), or partakes of a reflex character (chorea gravidarum and after injury).

3. *Endocarditis exists in the large majority of cases of*

chorea; and is either pre-choreic where the rheumatic influence has to be accused, *or post-choreic*, where we assume it to be due to irregularity in the action of the cardiac muscle; *but endocarditis cannot be considered a cause of chorea.*

4. Chorea is owing to hyperæmia of the territory of the middle cerebral artery, and more particularly of the corpora striata. In cases which end favourably, this hyperæmia does not proceed to rupture of the bloodvessels; but in fatal cases effusion of blood and consequent injury to the surrounding tissue take place.

5. A similar affection of the spinal cord, more particularly in the region of the posterior horns, is generally associated with the cerebral changes; and where mental symptoms have been prominent during life, it is probable that the cineritious structure of the convolutions of the brain has also been in a state of hyperæmia.

CHAPTER XI.

OTHER STRUCTURAL DISEASES OF THE NERVOUS CENTRES.

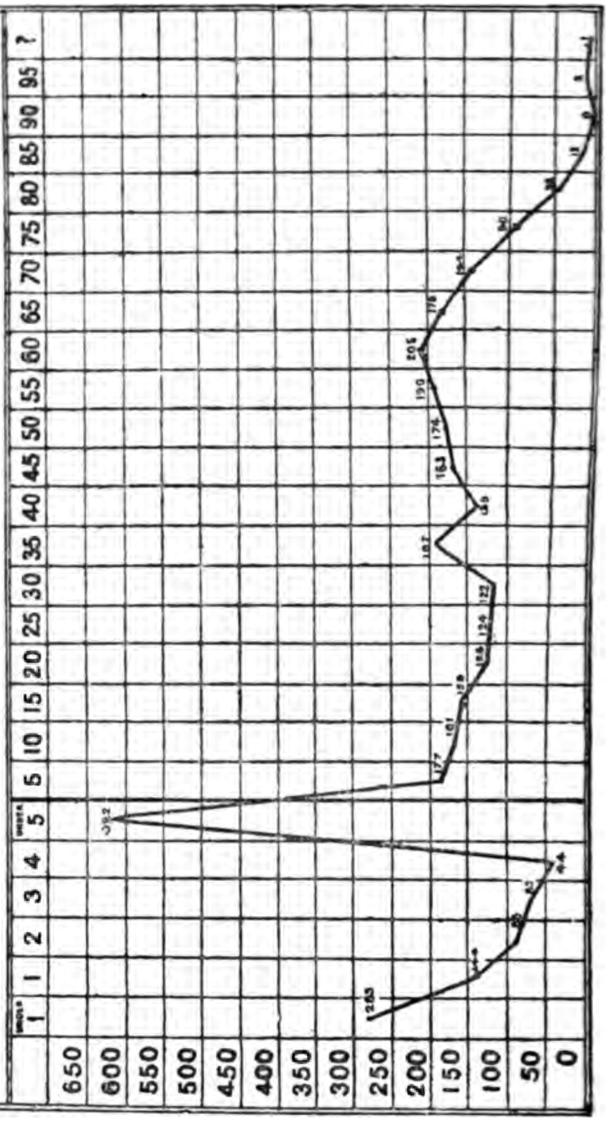
THE information given by the Registrar-General under the heading of 'Disease of the brain,' etc., does not help us in gaining an insight into the mortality from the several affections which I shall discuss in this chapter, as not only various maladies of the nervous centres, but also fatal diseases of the eyes and ears are included in this rubric. Nevertheless I have thought it better not to omit the details given, in order to render my work as complete as possible: and it should be understood that in doing so I have no wish to draw any general conclusions from the numbers which I shall now detail.

The diseases included in this rubric have on the whole shown a tolerably steady rise in mortality during the period over which the investigation extends. It is seen from the following table that a minimum of four per cent. occurred in the first lustrum, after which a decided increase took place in the second, a slight one in the third, and another considerable one in the fourth. The fifth lustrum showed a fall, but the sixth attained a maximum of nine per cent.

Periods of five years	Deaths from Brain Disease, &c.	Percentage of Nervous Diseases	Percentage of all diseases
1838–42	8,685	4·14	·50
1843–46	vacat	—	—
1847–51	15,079	6·28	·72
1852–56	17,449	6·80	·84
1857–61	22,871	8·49	1·05
1862–66	20,988	7·19	·87
1867–71	27,820	9·00	1·12

DIAGRAM N

Showing the influence of **AGE** on the Mortality from **BRAIN DISEASE** in England and Wales in 1847



The influence of age appears to be very marked on the diseases comprehended in this rubric. Like convulsions they are most fatal in the first year. The curve in Diagram N is seen to descend after that until the fifth, and reaches its summit in the first lustrum of life. After that it descends, rises again somewhat at thirty-five and forty, and falls more decidedly at fifty-five. At sixty there is another small rise, but after that the fall is steady, and at ninety there is nothing left.

The influence of sex is also considerable, for the following table shows throughout a larger death-rate for males than for females, and this difference between the two sexes has of late years become more marked; for while the mortality of males shows a decided rise in each lustrum, there was for females a fall in the second lustrum, a rise in the third and fourth, and the fifth has been identical with its predecessor.

Periods of five years	Males	Per cent.	Females	Per cent.
1847–51	8,239	3·42	6,840	2·85
1852–56	9,743	3·77	7,706	2·65
1857–61	12,815	4·71	10,056	3·70
1862–66	14,802	5·07	11,086	3·78
1867–71	16,060	5·20	11,665	3·78

I now proceed to consider the following structural diseases of the spinal cord and brain, which have not found place in the preceding chapters:—

1. Progressive locomotor ataxy.
2. Progressive muscular atrophy.
3. Pseudo-muscular hypertrophy.
4. Disseminated insular sclerosis.
5. Hypertrophy of the brain.
6. Atrophy of the brain.
7. Tumours of the brain.
8. Syphilitic affections of the nervous system.

1. *Progressive Locomotor Ataxy.*

Tabes dorsalis is first spoken of in the works of Hippocrates, and was by the father of medicine believed to arise from excesses

in sexual intercourse, the chief symptoms of the disease being spermatorrhœa, marasmus, and hectic fever. This meaning of the term, however, has gradually changed, and those authors who wrote on tabes in the first decennia of this century, understood by it atrophy of the posterior portion of the spinal cord, brought on, not merely by sexual exhaustion, but also by exposure to wet, rheumatism, gout, and other causes, the chief symptom being a peculiar form of paraplegia. The disorder was chiefly investigated by English and German physicians, such as Abercrombie, Hufeland, Steinthal, Romberg, and others. Their descriptions, although in some instances most eloquent, were, however, to a certain extent, wanting in accuracy, inasmuch as several different affections of the cord were comprehended under the name of tabes, and a clear distinction was not drawn between tabes and paraplegia. It was only after a more careful clinical study of the symptoms had been made, and after pathological anatomy, aided by the microscope, had stepped in, that a peculiar disease of itself, and one characterised by uniform structural lesions, could take its place in our nosological system. The chief credit of the anatomical investigations is due to Professors Virchow, Türck, Rokitansky, and Leyden, and in this country to Sir William Gull and Dr. Lockhart Clarke, who have shown that, in well-marked cases of tabes, an actual waste of the posterior columns of the spinal cord takes place, together with the formation of amyloid corpuscles, and considerable proliferation of connective tissue.

The first who drew a distinction between this disease and paralysis was Dr. Todd, who stated that two kinds of paralysis might be noticed in the lower extremities: the one consisting simply in the impairment or loss of voluntary motion; the other distinguished by a diminution or total absence of the power of co-ordinating movements. In the latter form, while considerable muscular power remained, the patient found great difficulty in walking, and the gait was so tottering and uncertain that his centre of gravity was easily displaced. In these few words we have a good description of the symptom of *ataxy*,[1] upon which so much stress has been laid by French physicians. The term 'ataxy' is as old as that of 'tabes,' for it also

[1] From τάξις, *order*, and privative alpha (want of order)

originated with Hippocrates; and it has likewise entirely changed its meaning in the course of time. Some authors have applied it to chorea, others to fevers, others to various nervous disorders. At present, however, we understand by ataxy, not a disease of itself, but merely a symptom to which various disorders may give rise, and which essentially consists of a want of co-ordination of voluntary movements, and a tendency on the part of the patient to lose his balance, but without actual loss of power, and apart from tremor, chorea, and paralysis. This symptom may be observed in disease of the cerebellum, and in poisoning by alcohol, lead, and mercury; but it is more especially connected with that disease which has been long familiar to us as tabes. The best clinical study of this symptom we owe to M. Duchenne de Boulogne, who, in 1858, described what he thought to be an entirely new disease, which he called 'progressive locomotor ataxy,' and which he believed to be a functional disorder of the cerebellum. His apparent discovery was hailed as a real one in France, and Professor Trousseau actually proposed to call the new stranger 'Duchenne's disease;' but on looking more closely into the matter, it was discovered that Duchenne's description was altogether applicable to our old friend, tabes. This is not said in disparagement of the great ability and originality of M. Duchenne's researches, which were perhaps more strikingly displayed in this case just on account of his being unacquainted with the previous literature on the subject; yet, if I thought it desirable to attach a proper name to this affection, I should prefer calling it 'Todd's disease,' as Todd first drew the distinction between ataxy and paralysis, eleven years previous to Duchenne. But the best plan is, perhaps, merely to drop the term 'tabes,' as being too vague, and to call the disease under consideration progressive locomotor ataxy, or wasting of the posterior columns of the spinal cord.

The following are the anatomical features of the disease:—

The vertebræ and the vertebral canal are healthy, but the sac of the dura mater often contains a somewhat considerable amount of clear or turbid liquid. The membranes themselves may be normal, but in some cases the *posterior* part of the dura has been found thickened, and adherent to the pia by thin false membranes, the *anterior* part of the dura being unaffected.

The posterior part of the pia is less transparent than it should be, and presents a yellowish or milky appearance. It often adheres so firmly to the substance of the posterior columns that it cannot be separated from them without tearing off some portions of the medullary matter. These changes have, however, only been noticed in about one-half of the cases examined, and we must, therefore, consider them rather as incidental, than as pathognomonic appearances. The latter are found in the cord itself, which shows, in its posterior columns, a peculiar grey coloration which is not superficial, but embraces their entire depth, and constitutes the characteristic anatomical feature of the disease, being always connected with a definite alteration of the intimate structure of the cord. In cases where the pia is opaque, it is necessary to remove it to show the grey coloration, but where that membrane is transparent, it becomes visible immediately upon the removal of the dura. We then see, instead of the white matter of the posterior columns, either one or two grey bands proceeding from the lower end of the cord to the middle of its dorsal portion, and occupying the whole space between the opposite insertions of the posterior roots. As we proceed higher up, these bands become narrower, and often separate into smaller stripes, which run up to the calamus scriptorius and the floor of the fourth ventricle.

The grey colour of the posterior columns is sometimes uniform throughout, in other cases it is dark in the median line, and light laterally. Occasionally the grey merges into amber, pink, or reddish yellow, according to the stages of the degenerative process. Generally, however, it is so similar to that of the healthy grey matter, that Olivier believed the whole process to be one of hypertrophy of the latter substance. Laterally the grey coloration is mostly limited by the posterior horns, and centrally by the commissure. This commissure and the central part of the grey matter are sometimes also affected, and in far advanced cases the disease may extend to the lateral columns; while the anterior horns, columns, and roots have always been found healthy.

The shape of the cord is altered, and being flattened from before backwards, it would seem at first sight to be actually

enlarged. Such, however, is not the case, for the flattening results from the diminution of the bulk of the posterior columns. Where the disease has been severe, they may be entirely gone, being replaced by a thin band of connective tissue. The consistence of the grey matter and of the antero-lateral columns is normal, which may also be the case with the posterior columns; occasionally, however, these latter are found softened, and even semi-fluid.

The posterior roots are, in most cases, similarly affected to the posterior columns. Sometimes the whole substance of the root is in a state of degeneration, while in other instances grey stripes alternate with healthy white bands. The lower roots are generally more affected than the upper ones. This affection of the roots is secondary to that of the columns, because in a certain number of cases the columns have been found diseased, while the roots were healthy, and because in other instances where both were diseased, the columns were in a far more advanced stage of degeneration than the roots. Moreover, it is found that in most cases, although columns and roots may be diseased at the lower part of the cord, yet, further upwards, the columns may show extensive disease, while the roots appear healthy. In no case has there been atrophy of posterior roots without simultaneous atrophy of the columns.

In the upper portion of the cerebro-spinal axis the disease is less severe than in the lower part. The calamus scriptorius occasionally shows traces of it, but the cerebellum which has in all cases been examined with the greatest care, has always been found healthy. This latter circumstance has caused considerable disappointment to certain pathologists who concluded *à priori*, from physiological premises, that structural changes of the cerebellum *must* be found in progressive ataxy. Such, however, is not the case, and this fact well illustrates how necessary it is to be cautious in the application of physiological theories to pathological processes.

The cerebral nerves may likewise show structural alterations. The optic nerves have been found softened or entirely destroyed, only a few fibrous strings being seen in place of nervous matter. The ulceration may spread to the chiasma, but stops short at the corpora geniculata. In the retina it proceeds from the

papillæ to the periphery of that membrane. The other cerebral nerves are rarely affected. In one case the olfactory nerves, although apparently healthy, were, on being examined with the microscope, seen to be almost smothered by amyloid corpuscles. The motor oculi, the hypoglossus, and the vagus have occasionally been found wasted. These results correspond with the symptoms observed during life; for while the cord and the optic nerves, when once thoroughly altered, are generally permanently disabled, the symptoms referable to the other cerebral nerves almost always disappear after a short time.

In a case which was some time ago under my care in the hospital, there was total deafness from disease in the semicircular canals. The patient recovered completely from all symptoms of ataxy, except the deafness; and it is therefore probable that in this case wasting of the auditory nerve would eventually be discovered.

The microscopic examination of hardened specimens shows that, whereas a section of healthy nervous matter taken from the anterior columns is dark, one from the diseased posterior columns is transparent. This results from the fact that in the anterior columns healthy nerve-tubes are crowded together, while in the posterior columns most of these have been destroyed by the disease, and are replaced by a clear and nearly homogeneous mass, which contains a number of small granules and connective tissue.

The nerve-tubes, if not entirely destroyed, appear granular, varicose, more narrow than usual, and are nearly or entirely devoid of myeline and the cylinder axis. There is also proliferation of connective tissue; the capillary vessels show thickening of the adventitia and appear surrounded by oil-globules. A large number of amyloid corpuscles, presenting the well-known mother-of-pearl appearance, are likewise met with. They are most numerous along the course of the blood-vessels, and abound chiefly where the degeneration is not far advanced, while they are less frequent where the nervous matter is quite destroyed. The lesions of ataxy are therefore destruction of nervous matter, proliferation of connective tissue, degeneration of blood-vessels, and formation of corpora amylacea and oil-globules. It resembles both chronic inflammation and simple

atrophy, but neither of them altogether, and should therefore be looked upon as a lesion '*sui generis*.'

Duchenne has distinguished three stages of the disease, but these are by no means always so well defined as this author would lead us to believe. The first stage is marked by certain affections of the cerebral nerves, pains of a peculiar character, and diminution of sexual power; it generally lasts from four to five years, sometimes much shorter, and in other cases longer. In the second stage the symptom of ataxy supervenes, together with loss of sensibility; this may last ten years and more. In the third stage the symptoms of the first and second stage become more severe; complications, such as paralysis and spasms, supervene, and death results from exhaustion, or from intercurrent diseases.

The commencement of progressive ataxy is either slow or subacute. One or more of the cerebral nerves are generally the first to suffer, those most frequently affected being the optic, and the third, fourth, and sixth pairs. The chief symptoms are, therefore, amblyopia, double vision, strabismus, and ptosis. Sometimes there is even treble vision, or two images may be observed by one eye while the other is closed—symptoms which have not yet found a satisfactory physiological explanation. The ophthalmoscopic examination of the fundus oculi shows, at first, symptoms of congestion; the vessels are diseased, and the whole fundus has a violet colour. Strabismus and double vision have the tendency to disappear within a few months, with or without treatment; ptosis is liable to continue much longer, and amblyopia almost always, in the course of time, merges into amaurosis. The ophthalmoscope then shows signs of atrophy of the retina; the diameter of the blood-vessels is diminished, the disc is of a greyish or mother-of-pearl hue and excavated, and a white circle is seen at its margin.

The other cerebral nerves may also show signs of paralysis, with the only exception of the olfactory; there may be loss of taste, deafness, difficulty of mastication, dysphagia, and numbness or loss of sensation of the face, lips, tongue, and gums. These latter symptoms are, however, comparatively rare.

Most diseases of the cord are accompanied by *pain*, but progressive ataxy more frequently than any other. Pain of a

peculiar character constitutes, indeed, one of the most distressing symptoms of this affection. It is short, sharp, and sudden, similar to electric shocks. After a second or two the pain is gone, and the patient has a short interval of rest; but soon there is another pang, and this may go on for two or three days consecutively, after which there is a free interval of a few weeks or even months. The pain often begins in the feet, then migrates about the body, sparing only the head, and finally settles in one of the legs, from where, as the disease advances, it gradually proceeds upwards. During the attacks neither swelling nor redness is perceptible in the parts affected, but after some time considerable hyperæsthesia sets in, so that the patient is exceedingly sensitive to touch or even a slight draught of air. In other cases there is no hyperæsthesia, but numbness, and strong pressure relieves the pain. If the eyes are attacked, a flow of tears, heat, and dilatation of the pupils are caused; if the bladder is invaded, catarrh of that organ may be produced.

As time wears on, the pain generally increases in severity, appears at shorter intervals, and lasts much longer. It is most liable to come on when sudden atmospheric changes occur, after exposure to wet, after excesses in walking, drinking, or sexual intercourse, and from indigestion. The patients generally dread winter. As spring advances they frequently improve, and this is often believed to be due to the remedies which happen to be employed about that time. Some patients consider themselves weather-glasses, as they are by an increase in the severity of the pain generally able to predict an impending change.

Spermatorrhœa is another important symptom, but it is wanting in a number of cases. When present, it seems to accelerate the progress of the disease. Emissions occur first at night, and with erections; after a time they likewise occur in the daytime, and without erections, more especially on voiding the bowels, and impotence is the final result. In exceptional cases the disease is ushered in by priapism and satyriasis. Eisenmann has recorded a case in which these latter symptoms continued more or less for thirty years, and were only relieved by large doses of opium.

The bladder and rectum generally suffer at a somewhat

early stage of the complaint. Constipation is the rule, while involuntary fæcal discharges are rare. The bladder is not completely emptied, and the urine passes tardily. Incontinence may also be present.

After a time, other symptoms come on, either suddenly or gradually, by which the second period of the complaint is marked. The most important of these, and from which the disease has received its name, is the locomotor ataxy, or loss of co-ordination. Volition loses its influence over the muscles, which, although still possessed of great intrinsic force, are nevertheless unable to execute complex movements, or preserve the equilibrium of the body in its erect position.

The ataxy generally begins in the lower extremities. The patient notices an awkwardness in his movements when he walks in the dark, or in the morning while he is dressing. He soon takes to a stick when out of doors, but even with such aid he finds that he has to make considerable efforts to prevent himself from falling. In order to appreciate the degree of ataxy which may be present, we must examine the patient in all positions, whilst standing, walking, and lying down. If he is told to stand with both feet close together, he can seldom keep his balance. He staggers from one side to the other, and manœuvres desperately with his arms, almost like a rope-dancer; but unless supported, he would go down at last. If told to stand with his eyes closed, he has to struggle very hard to maintain himself. But this alone is not sufficient to enable us to diagnose progressive ataxy, because the same symptoms may be found in convalescents from acute diseases, in certain cerebral affections, or in persons who are reduced by bad living, and in a weakly condition.

The ataxy becomes much more apparent if the patient attempts to walk. He throws the legs forward with a jerking motion, and puts the feet down with great force. In turning round, he is especially awkward. At the commencement of the affection he can still walk a considerable distance, and feels the difficulty chiefly on first starting, or changing his direction; but as the disease advances, walking becomes almost, or quite, impossible. He is still able to make powerful muscular efforts, and flexors as well as extensors feel hard and contracted; yet he does not succeed

in doing that which other persons accomplish without an effort. At first the ataxy is more striking in the pelvic and femoral muscles. But after a time it becomes also apparent in the leg and foot. The sole seems continually to search for support, one leg is crossed over the other, or jerked about in a disorderly manner, without the slightest intent or purpose. All efforts of the will to check these movements are ineffectual and only serve to increase them. The patient soon becomes exhausted by the expenditure of so large an amount of muscular force, and is glad to get back to his couch.

While laying down, the patient is able to move the legs in all directions, showing that there is no paralysis; but these movements are often abrupt and sudden. In a somewhat advanced stage of the disease no graceful or easy movement is possible: he does not know what force to use, or where to stop, and he cannot continue a movement for any length of time. The difference between this condition and paralysis is very striking. The atactic patient has a great deal of muscular power, and is able to make efforts to do what he is requested to do, but he does not know how to set about it, and expends his force in vain. In the paralytic, on the contrary, the power is lost, or greatly diminished; he cannot move, or if he succeeds in doing so, it is a feeble motion, although one not devoid of purpose.

In the upper extremities ataxy is not so well marked, nor so frequent as in the lower ones; and it mostly appears only at a later period of the disease. If the patient be requested to touch his nose with the tip of the forefinger, to pick up a pin or a piece of money, to describe a circle in the air, &c., the ataxy becomes apparent, more especially if the eyes be closed at the same time. He cannot write in a straight line, and is awkward in dressing and feeding himself. The muscles of the face are only rarely affected; sometimes, however, there is facial spasm and difficulty of articulation.

Sensibility always suffers at this stage of the complaint. The patient experiences a feeling of numbness or heaviness in a limb or part of a limb, which may exist without any loss of cutaneous sensation. It generally commences in the toes, or the soles of the feet, and from there gradually spreads upwards

to the abdomen and the chest, which feel as if constricted by a circular band, a net, or a tight string. Where this feeling invades the chest, dyspnœa is also present. In the upper extremities the numbness is generally confined to the third and little finger, and only seldom spreads higher up. Numbness in the legs and feet is one of the most constant symptoms of the disease, and if absent, must make us doubtful whether the case is really one of ataxy or not; and when it diminishes or disappears, we may say with certainty that an improvement has taken place.

Cutaneous anæsthesia is a frequent, but by no means constant symptom of ataxy, and generally appears only at a somewhat later period of the disease. We may observe all the different forms of anæsthesia, viz.—loss of the sensation of pain, or analgesia; loss of the sense of touch, which is anæsthesia properly so called; and loss of the sense of locality, temperature, and pressure.

Sensibility to pain may be diminished or entirely gone. Neither pricking nor pinching are felt, or instead of a sharp sensation, a dull kind of touch may be perceived. Cruveilhier mentions the case of a man who had fractured the leg, and neither at the time of the accident, nor afterwards, had felt any pain whatever. The sensibility to galvanism and faradism is also diminished, as the patient is able to bear the application of a powerful current without inconvenience. The sense of touch is dull or gone. The patient cannot distinguish the nature of an object by touching it with his fingers, but has to be assisted by his eyes, and this is not confined to the upper or lower extremities, but may extend to the neck, tongue, and soft palate. A common symptom is tardy sensation, so that when touched, the patient only feels it five or ten seconds afterwards. If the soles of the feet are tickled, there are scarcely any or no reflex movements, and the perception of tickling is dull or absent.

The sense of locality is often wanting, so that the patient, if touched in a particular part of the body, cannot tell you where he is touched. The distance at which two separate sensations are perceived as such, is increased, as may be shown by examining him with Weber's pair of compasses. In the legs, where the normal distance for two separate sensations is an inch

and a half, the patient is sometimes altogether unable to feel them as such, while in the face and fingers he may still be able to do so.

The *sense of temperature* is only rarely deficient. We are generally able to distinguish with certainty a difference of one, or half a degree, and this faculty may remain after all other kinds of sensation have vanished. M. Topinard has recorded such a case which was under Trousseau's care in the Hôtel-Dieu. The patient had double amaurosis, absolute anæsthesia as regards touch, pain, and locality, ataxy of motion to such an extent that he had been unable to get out of bed for two months; he had completely lost his muscular sense, and had only the sensation of heat and cold to tell him that he still had his limbs. Sometimes his legs would be jerked out of bed by spasms which he did not feel, and which, being blind, he could not see. Then, after a time, a sensation of cold would creep upon him, and the poor fellow would ask whether anything was the matter with his legs. This patient existed, as it were, only by his memory, as he had lost the consciousness of his body. But even the sense of temperature may be wanting. Leyden mentions the case of a man who prepared a warm bath for himself, and not being able to distinguish between heat and cold, he made it too hot, and was severely scalded on going into it.

The sense of pressure, which is only a modification of that of touch, resides in the nerves of the skin, cellular tissue, muscles, and periosteum. A sensation of pressure is produced by weights resting on certain parts of the body, more especially on bones. M. Eigenbrod, who had studied the changes which this sense undergoes in various affections, has found that there is a considerable diminution of it in ataxy. Persons in good health are generally able to distinguish a weight of thirty from one which is only twenty-nine pounds, but the atactic patient loses this faculty to a great extent. If the sense of pressure in the soles of the feet is artificially diminished by the application of ice or chloroform, the gait becomes tottering: and it is, therefore, permitted to suppose that the uncertain walk of atactic patients is partly owing to the diminution of this sense, a firm gait being only possible where there is a proper sensation of resistance offered by the ground on which we walk.

In the third period of the disease we observe all the symptoms which are present in the second, only in a more marked degree. Sensation becomes more impaired, the ataxy more striking, and muscular force, which was previously intact, begins to fail. The patient cannot grasp anything with sufficient force; he cannot offer any resistance to movements imparted to his limbs by others; he has difficulty in raising his legs when in bed; and if he attempts to walk, the feet drag on the ground. The muscles waste away and undergo fatty degeneration. Spasms often supervene, which are most troublesome at night and when the weather is damp. Amblyopia merges into amaurosis; there is paralysis of the bladder, incontinence or retention of urine, involuntary evacuation of fæces, and at last decubitus on the sacrum, unless great care is taken to prevent it. The bones and joints also become affected; fractures are produced without injury while the patient is in bed, or standing, and in a similar way the hip-joint and other articulations may become dislocated, without any apparent cause. Death ultimately takes place from bedsores or inflammation of the bladder and kidneys, or from intercurrent diseases, such as bronchitis, pneumonia, and phthisis.

A curious symptom remains to be mentioned—viz., the state of mind of such patients. Unless the pain be severe, they are mostly, if not happy, at all events resigned to their fate. They do not complain much, and are inclined to think lightly of their affection. In this respect progressive ataxy resembles pulmonary consumption, in which the mind is also often composed and cheerful, while in diseases of the brain and the liver there is almost always great depression or irritability, or both combined.

Ataxy rarely occurs before thirty years of age, being more frequent after forty; and it is seldom seen after sixty. The male sex is more liable to it than the female. Romberg says that scarcely one-eighth of the cases are females. In the observations recorded by French authors and tabulated by M. Topinard, the proportion is of one female to four males. Of thirty-four cases which I have seen, only one occurred in a woman. Persons who are much exposed to cold, damp, and fatigue, are more liable to it than others. Several of my patients were commercial travellers, who had to be continually on the move. In another case the cause of the illness was attributed by the patient to his

having been obliged, after a ball in which he had taken active part, to walk home in thin boots in a pelting rain, having been unable to get a conveyance. In this case the symptoms supervened with great rapidity.

Soldiers are also very liable to it. 'The malady is rife,' says Romberg, 'when the strength is much taxed by continued standing in a bent posture, by forced marches, and the catarrhal influences of wet bivouacs, followed by drunkenness and debauchery, as is so often the case in campaigns; and this is the reason why tabes dorsalis was so frequent during the first decennia following the great French wars of the present century.'

Accidents are unquestionably a fruitful cause of ataxy; and a fall from a horse seems to bring it on more particularly. Sometimes two or three years elapse between the accident and the outbreak of the disease. Ataxy is more liable to come on in autumn and winter than in spring and summer. Most patients improve during the summer months; and we must, therefore, not be hasty in ascribing any beneficial results which may appear during that season to the treatment employed; while, if they get better in winter, there is more probability of the remedies used having been of actual service.

This malady is believed to be mostly owing to sexual excesses, but experience goes far to prove that such excesses tend to produce cerebral rather than spinal affections, and it is erroneous to look upon them as the chief cause of ataxy.

2. *Progressive muscular atrophy.*

This disease, which was first described by Duchenne and Aran (1849–50), is characterised by a gradual wasting of the voluntary muscles of, and proportionate loss of motor power in, the extremities. Friedreich,[1] to whom we owe the most careful description of the morbid process which takes place in the muscular tissue, has described it as *progressive chronic polymyositis*. There is at first proliferation of the interstitial connective tissue of the internal perimysium, between the several primitive bundles. The

[1] 'Ueber progressive Muskelatrophie, und wahre und falsche Muskelhypertrophie.' Berlin, 1873.

muscular corpuscles are at the same time seen to be swollen and increased, their nuclei proliferated, and the transverse stripes appear cloudy and granular. In some cases there is hypertrophy of the primitive fibres, and dichotomic or trichotomic division of the same. As the connective tissue continues to proliferate, the muscular fibres perish, either by simple wasting, or after previous division, or by fatty and lardaceous degeneration. Ultimately the muscle is found to have undergone cirrhosis or fibrous degeneration, and then has the appearance of a tough thin chord or a tendinous membrane, which only shows a few remaining insular patches of reddish muscular tissue. There is consequently considerable decrease of bulk. Occasionally, however, myositis may become complicated, either at an early stage or towards the end, with *diffuse lipomatosis*, which never commences in the muscles themselves, but always in the interstitial connective tissue, when this has commenced to proliferate. Fat-cells originate from the connective-tissue-corpuscles, which are seen to be filled with small globules, and these latter gradually conglomerate so as to form regular drops of fat. When this occurs, the bulk of the muscle is again augmented, and may even increase beyond its original size. Within this fatty mass, however, the originally fibrous structure of the muscle and the arrangement of the bundles may still be recognised by the peculiar arrangement of the different layers of fat.

Chronic polymyositis and diffuse secondary lipomatosis are, however, not the only anatomical changes which are found in this disease, for certain morbid alterations of the nervous system are likewise, if not invariably, at least commonly, present. Cruveilhier was the first to draw attention to these, and discovered wasting of the anterior roots of the spinal nerves in the celebrated case of the rope-dancer Lecomte. Such lesions have since then been discovered by many observers, while others again have found the nerve-roots perfectly healthy. In Lecomte's case the cord itself was not diseased, but in other cases softening of various parts of that organ was discovered. Luys first directed attention to certain alterations of the grey matter in the centre of the cord, having found the ganglionic cells of the anterior horns wasted, and replaced by granular

masses containing oil-globules. This was confirmed by the observations of Dr. Lockhart Clarke,[1] who noticed in six cases dilatation and hyperæmia of the blood-vessels, and granular disintegration of the ganglionic cells, resulting from an irritative process, or induration with hyperplasia of connective tissue, and wasting of nervous structures. Sir William Gull has described a similar condition of the grey matter, with great dilatation of the spinal canal between the fifth cervical and third dorsal vertebræ, the vacuum being filled with serous liquid, while the white columns and the nerve-roots were found normal. Other observers have noticed atrophy in the antero-lateral and posterior columns, the posterior cornua, the posterior roots, the intervertebral ganglia, the cervical sympathetic nerve (Schneevogt and Jaccoud), the peripheral nerve-trunks, and the intra-muscular nerve-fibres, which latter are found to have undergone peri-neuritis and interstitial chronic neuritis.

Seeing these extensive changes found in the muscles as well as in the nervous structures, the question has naturally arisen and been much discussed whether the origin of the disease is myopathic or neuropathic. Friedreich, who has with great ability contended for the myopathic theory, is of opinion that progressive muscular atrophy commences as primary myositis, and may in its turn lead to secondary changes in the nervous system, which consist of neuritis affecting first the intra-muscular nerves, afterwards the nerve-trunks and the spinal roots, and ultimately the cord itself. This inflammation may, according to him, become arrested at any part; and its further propagation within the nerve-substance essentially depends upon the more or less active character of the myositis, which has to be looked upon as the source of irritation. The degenerative processes occurring in the peripheral nerves and ganglionic cells of the anterior horns of the cord would, therefore, have to be considered as simple consequences of disturbed motor function and ascending neuritis.

It is difficult to explain by this theory why the nerve-roots and peripheral nerves have so often been found healthy when the anterior horns were diseased; but it is more especially the

[1] 'Medico-Chirurgical Transactions;' London, 1868, p. 249.

association of progressive muscular atrophy with progressive bulbar paralysis, which occurs in many cases, that militates most strongly against the myopathic theory of the complaint. Nobody now looks upon progressive bulbar paralysis as a muscular disease, for it has been conclusively shown to arise from wasting of the ganglionic cells of the motor nuclei in the rhomboid fossa; and as towards the end of progressive muscular atrophy the symptoms of bulbar paralysis frequently become developed, it is reasonable to assume that the disease is then spreading from the grey matter of the spinal cord to the medulla oblongata. Again, the later stages of locomotor ataxy may be combined with complete wasting of the muscles, when the disease has extended from the posterior columns right through the substance of the cord to the anterior horns of the grey matter. The symptoms of all these diseases are therefore seen to depend upon the areas of the cord and medulla which are affected in the first instance, and upon those which are afterwards involved in the morbid process.

Progressive muscular atrophy is thus seen to be a disease of the spinal cord; and chiefly of the ganglionic cells of the anterior horns of the grey matter, from where the motor roots emerge, and which preside over the nutrition of the muscles. The number of wasted muscles is fairly proportionate to the degree of atrophy which is found in the anterior cornua. The nature of the process is very similar to that which takes place in progressive locomotor ataxy—viz., grey degeneration of the nervous matter, increase and thickening of the connective tissue and fatty degeneration of the nerve-tubes; and only the seat of the disease differs, being in the one instance the posterior columns and in the other the anterior cornua. It also closely resembles the sclerosis which is found in the lateral columns of the cord, after attacks of apoplexy; and finally a form of disease which we shall presently consider under the heading of disseminated insular sclerosis of the brain and spinal cord.

In progressive muscular atrophy the sclerosis proceeds in a peculiar manner, affecting some ganglionic cells more, and others less, completely, some sooner and others later, while some are entirely spared. This explains the peculiarity of the symptoms of the disease, in which muscles in different parts of

the body are to all appearance capriciously attacked; and yet there is the tendency that certain sets of muscles which are either co-ordinated in their function, or symmetrical, suffer at the same time. There is nothing specific in the lesion, for there may be ordinary grey degeneration, or sclerosing myelitis, Clarke's granular disintegration, or pigmentary degeneration of the cells, but the ultimate result is always the same—viz., atrophy of the ganglionic cells, which perish one after the other. Friedreich, who considers the seat of the disease to be exclusively in the muscular tissue, thinks that it is caused by a nutritive and formative debility of the muscular tissue, which leads to diminished powers of resistance, and creates a tendency to irritative and degenerative disturbances of nutrition. Those, on the other hand, who look upon it as a disease of the cord find the cause of it in the neuropathic constitution, which in this instance manifests itself by debility of the ganglionic cells of the anterior horns, and which is evidenced by the simultaneous occurrence of such diseases as progressive bulbar paralysis, locomotor ataxy, and general paralysis of the insane.

That there is a constitutional tendency to the production of this complaint seems evidenced by the fact that a decided hereditary influence has been shown to exist in a considerable number of cases. Hemptenmacher[1] has given the details of a series of cases in which the males of three different families, which descended from a couple living 150 years ago, were subject to the complaint, and where the unaffected females transferred the germ of the disease to their descendants; and Trousseau has mentioned a family in which the great-grandfather, grandfather, father, and son suffered from this affection.

Adult males are particularly liable to the disease, the proportion being of about five men to one woman. This is probably to a great extent owing to the circumstance that men have harder muscular work to do than women, and that they are also more liable to accidents. The complaint is, however, not unfrequent in childhood, where the hereditary tendency is all-important. Accidental causes, which seem to give rise to

[1] 'De aetiologia atrophiæ muscularis progressivæ.' Berlin, 1862.

its outbreak, are certain acute diseases, such as measles, rheumatic and typhoid fever, and the puerperal state. Over-exertion of the muscles can be traced in another set of cases, which occur chiefly amongst the working classes, in males, and in which the right hand and shoulder are more affected than the left. In sixty-nine cases collected by Dr. Roberts this was the case in twenty-five.

Friedreich has shown that out of 146 cases the disease commenced in 111 in the upper extremities, in 27 in the lower limbs, and in 8 in the muscles of the loins. The muscle which is generally first affected is, according to Eulenburg,[1] the first external interosseous of the right hand, as shown by the following experiment:—If the thumb is adducted, and the second, third, and fourth finger are fixed, the patient experiences some difficulty in moving the extended first finger towards the radial side of the hand; and where the disease begins in one upper extremity, a great difference is noticed in this respect between the affected and the healthy side. Other observers, myself included, have seen the disease to commence in the ball of the thumb, affecting more particularly the opponens and adductor pollicis, while extensors, flexors, and abductors remained healthy. The muscles of the forearm and arm are generally spared in the commencement of the affection, which makes a sort of jump from the muscles of the fingers to the deltoid muscle, which in some cases has even been the very first attacked. The trapezius and serratus follow, and this causes displacement of the shoulder-blade, which is twisted round its axis; the upper angle of the bone is pulled down by the weight of the upper extremity, and the lower angle then becomes raised and withdrawn from the chest-wall. From the shoulder the disease spreads towards the arm, destroying the biceps and triceps; the patient can then no longer raise the arm or bend the elbow-joint, and has great difficulty in dressing and feeding; and although he learns in time to manœuvre cleverly so as to compel muscles which have escaped destruction to do the work of those that are wasted, he nevertheless becomes at last completely helpless.

[1] 'Vasomotorisch-trophische Neurosen.' In 'Ziemssen's Handbuch,' vol xii. part i. p. 114. Leipzig, 1877.

Contractions and deformities are also noticed in the hand, which assumes the shape of a bird's claw from wasting of both internal and external interossei; the thumb is abducted and extended in the first, but flexed in the second phalange. The shoulder-joint may become dislocated, the caput humeri being directed towards the coracoid process. This is more particularly seen in children.

Wasting of the muscles of the lower extremities causes great difficulty in walking, and particularly in going upstairs. The rectus femoris and the hamstring muscles are generally the first to suffer, and after them follow the adductors of the thigh and the gastrocnemius muscles. The pectoralis major is usually attacked about the same time, and the chest then appears shrunk, more especially beneath the collar-bones.

Where the disease begins in the lower extremities, the patients are mostly children, and it is then not real atrophy, but pseudo-hypertrophy of the muscles, which will be presently considered.

Farado-muscular contractility diminishes in the same ratio as the fibres waste; but as long as any such exist, they remain excitable to faradisation. The same may be said of galvanisation, but there is this peculiarity with the latter, that the motor nerves respond differently to the current in different portions of their course between the spinal cord and the muscles, so that they may be still excitable near the cord when their excitability is diminished or entirely gone in their more peripheral portions.

A curious symptom is the occurrence of fibrillary twitches in the wasting muscles, which indicate that the disease is advancing; when they cease, there is either improvement, or it is owing to total destruction of the suffering muscles. Regular cramp occurs likewise, especially towards the end, and when the patient is in bed. The adductors of the thighs are then often in a constant spasmodic motion, so that the knees keep knocking against one another, which prevents the patient from sleeping.

We rarely meet in this disease with any symptoms pointing to altered sensibility, but in some cases paroxysmal pains have been observed in the beginning, and anæsthesia towards the

end. The temperature may be at first increased, and later on diminished. Sometimes there is sudden ischæmia of the hands and fingers with subsequent hyperæmia, or profuse perspiration. The skin is often wasted, rough, and discoloured, and painful affections of the joints are not uncommon. The pupils are sometimes narrowed, and respond sluggishly to the action of light, most probably from affection of the cervical sympathetic nerve. The urine has now and then been found alkaline.

The large joints seem occasionally to offer a certain amount of resistance to the propagation of the disease, which, as we have seen, does not affect the muscles in a contiguous fashion. Thus atrophy of the muscles of the hand rarely, if ever, proceeds straight to the muscles of the forearm; and when it commences in the deltoid, the elbow-joint seems to prevent it from extending lower down. The muscles of the neck and head, as well as of the eyes, are generally spared; and the diaphragm and abdominal muscles suffer, if at all, only in the later periods of the disease.

The course of progressive muscular atrophy is generally slow, and protracted over a good many years. It is never, or hardly ever, cured, but its progress is sometimes arrested, and the patient may then live on for years in tolerable comfort, provided the disease has ceased to be active before too much loss of tissue has occurred. Death generally occurs from bronchitis, as the expiratory muscles suffer, and the expectoration of mucus becomes difficult or impossible; or from decubitus and blood-poisoning; or from the disease spreading to the medulla oblongata, and bulbar paralysis, with all its consequences, being developed. Wasting of the facial muscles, which causes the physiognomy to lose all expression, is a sure sign that the end is near.

3. *Pseudo-hypertrophy of the muscles.*

This disease, which is closely allied to progressive muscular atrophy, was first described by two Neapolitan physicians, Coste and Gioja, in 1838, and afterwards more fully by my colleague, Dr. Meryon, in 1852. In spite of these and numerous later

observations, the pathology of the affection, and more especially its relation to progressive muscular atrophy, are still somewhat obscure. Its principal feature is proliferation of the connective and adipose tissue, by which the circumference of the affected limbs is very much increased, and simultaneous wasting of the muscular fibres, which causes loss of motor power in the extremities.

The morbid process in the muscles is probably of an inflammatory nature, and begins with proliferation of the connective tissue from the internal perimysium and the adventitia of the small blood-vessels. Occasionally the process is arrested at this stage, but in the large majority of cases, the spindles and nuclei of the connective tissue are gradually changed into oil-globules; and interstitial and inter-fibrillary development of fat then becomes the essential feature. The nutrition of the muscles suffers considerably in consequence of this; and the primitive fibres waste away under the influence of pressure. Sometimes a few truly hypertrophied fibres are found in the neighbourhood of the wasted fibres, and are seen to have two or three times the diameter of healthy fibres. The muscular nuclei may be increased in number, and the fibres show a certain amount of granular cloudiness which disappears on adding acetic acid, signs which must be attributed to an irritative process in the muscular fibres themselves. To the naked eye the muscles appear pale, white, and shining, and are scarcely to be distinguished from the subcutaneous adipose tissue. Whether any alterations of the nervous centres are connected with this disease, is as yet undecided. The results of most necropsies have been negative, but in a few cases granular disintegration of certain portions of the cord, and proliferation of connective tissue in the sciatic, tibial, and peronæal nerves, has been discovered.

Friedreich has expressed the opinion that pseudo-hypertrophy of the muscles is only a form of progressive muscular atrophy, which is somewhat modified by occurring particularly during childhood, and showing somewhat greater intensity of the morbid predisposition. The fact that when children begin to suffer from this disease, they are soon disabled from moving about, and condemned to repose, as well as the circumstance

that their principal food, more particularly where the affection occurs in the lower classes, consists of articles which either are fatty or changed into fat in the system, seem to lend considerable support to this view. It is well known to farmers that the formation of fat in animals is powerfully promoted by a tender age, want of exercise, and fatty food; and all these circumstances are found combined in the pseudo-muscular hypertrophy of childhood.

Dr. Ord has shown that the invasion of the malady is accompanied by an increase of temperature in the legs, which is evidently dependent upon the inflammatory nature of the connective tissue proliferation by which the complaint is ushered in. In the later stages of it, the temperature is lowered, sometimes very much so (10 to 15°), and the diseased parts have either a pale or livid colour. The skin is dry and smooth, has a mottled appearance, and is somewhat attenuated by the excessive tension of the parts underneath.

As a rule the affection begins in the legs. The calves stand out conspicuously, 'like those of a practised pedestrian,' and yet the patient is unable to stand or walk properly. He keeps stumbling, has a waddling gait, and is easily exhausted. The thighs soon follow in their turn, and the disease gradually progresses to the buttocks, the muscles of the lumbar spine, and the abdomen. The legs are unnaturally far apart, while the feet are kept close together; the heels are drawn up, the knees and hip-joints are flexed, and the spine shows various forms of curvature. On measuring the limbs from time to time, a steady increase of their circumference will be noticed. They have a spongy, fatty feel, which is utterly unmuscular, and is more striking in the calves than in the thighs. As the disease progresses, the patient finds it difficult to sit down or rise from a sitting posture, and the aid of the hands and arms is requisitioned, which, however, often refuse the service demanded of them, since they are liable to suffer from ordinary atrophy of the muscular tissue. Fibrillary twitches are occasionally present, but difficult to distinguish on account of the layers of fat which cover the muscles. The farado- and galvano-muscular excitability is at first diminished, and later on completely lost; while the electro-muscular sensibility is

sometimes increased. Pain in the back and in the lower extremities is complained of, especially when the patient attempts to move; and this is relieved by rest. Numbness and a feeling of coldness may likewise be present, but anæsthesia is rare.

It is doubtful whether such children ever attain a great age; but pseudo-hypertrophy is certainly not so dangerous to life as progressive atrophy. In cases which have ended fatally, death was generally owing to certain affections of the respiratory organs, such as bronchitis, pneumonia, &c.

Apart from pseudo-hypertrophy, *real muscular hypertrophy* has been observed in a few exceptional cases. There is increase in the bulk of the muscles, and the power may be increased or normal. It seems always to occur in adults who have undergone considerable fatigues, such as soldiers during a campaign; and is usually confined to one arm, or one leg, these being two or even three inches larger in circumference than their fellows. In some cases the power appears increased on first beginning to carry out certain movements, but soon becomes exhausted. The muscles have the proper consistency, and obey the induced as well as the continuous current, well, or at least tolerably well. On harpooning pieces of the enlarged muscles, and submitting them to microscopical examination, the structure appears normal, except that the cylinders are much wider than usual. Thus a muscular cylinder taken from the deltoid in Auerbach's[1] case, showed the width to be double that of specimens from healthy muscles. There is no trace of myositis or lipomatosis. That fatigue should be easily induced in the hypertrophied muscle, may either be owing to the circumstance that the intra-muscular blood-vessels and nerves do not grow at the same ratio as the muscular fibres, or to the pressure of the swollen muscular tissue on the intra-muscular nerves. No case has as yet ended fatally.

4. *Disseminated insular sclerosis of the nervous centres, sclérose en plaques disséminées, Charcot's disease.*

This malady was first described by Cruveilhier, and afterwards studied by Türck, Rindfleisch, Zenker and Frommann;

[1] 'Virchow's Archiv,' vol. 53, p. 234.

but we owe almost all precise knowledge concerning its anatomy and symptoms to M. Charcot,[1] and I therefore propose calling it *Charcot's disease*, a term which will advantageously replace the very long name by which it has hitherto been designated. It affects not only the hemispheres, pons, and cerebellum, but also the medulla and spinal cord, in which grey and more or less irregular patches are discovered, which form a striking contrast to the healthy tissues in which they are embedded. They somewhat resemble the grey matter, but assume a pink colour by contact with the air, and they are then seen to contain numerous bloodvessels. The cineritious substance of the convolutions is rarely affected in this manner, the chief seat of the sclerosis being the central ganglia and the antero-lateral columns of the cord. Grey patches are likewise found in certain peripheral nerves, more particularly in the olfactory, optic, and fifth, and the anterior and posterior spinal nerve-roots.

Charcot distinguishes three different zones of these patches, viz., 1st, the peripheral zone, 2nd, that of transition, and 3rd, the central one. In the *peripheral zone* the trabeculæ of the neuroglia are considerably thickened, the nuclei more bulky and numerous; the nerve-tubes lie at a greater distance from one another, the cylinder of myeline being wasted, while the cylinder axis is either normal or hypertrophied. In the *second zone or that of transition*, the nerve-tubes have become even more attenuated; the myeline cylinder has quite disappeared, while the cylinder axis appears much enlarged. The trabeculæ of the neuroglia are more transparent, and now and then replaced by ordinary connective tissue. In the *third or central zone* the neuroglia has quite disappeared; the nuclei are less numerous and bulky, and are not so well coloured by carmine as usual; no myeline is seen in the alveolar spaces, and the cylinder axis is considerably reduced in size. Nevertheless, a certain number of axis cylinders persists in the otherwise completely altered tissue, which constitutes an essential feature of this form of sclerosis, as it is absent both in the sclerosis of the lateral columns which follows apoplexy, and in the sclerosis of the posterior columns, which is characteristic of progressive locomo-

[1] 'Leçons sur les maladies du système nerveux.' Paris, 1872, p. 168.

tor ataxy. The coats of the bloodvessels are thickened in the peripheral zone, and contain a larger number of nuclei than usual; in the more central portions of the patches these nuclei are still more numerous, and the coats so thickened that the caliber of the vessel is considerably reduced. Amylaceous bodies are found interspersed in the fibrillary tissue. Oil-globules which constitute the débris of the destroyed nerve-tubes are wanting in the central zone where the pathological process is finished, but occur plentifully in the more peripheral portions. This form of sclerosis is, therefore, anatomically speaking, a chronic interstitial myelo-encephalitis.

Charcot's disease affects women rather than men, and appears with preference in comparatively young persons, between 20 and 25 years of age. It rarely comes on after 30 years, and patients suffering from it do not as a rule reach the age of 40. The influence of wet and cold, prolonged anxiety, and certain acute diseases such as cholera and typhoid fever, seem occasionally to have given rise to it. Three different periods may be distinguished in its course, which I shall now proceed to describe.

In the first period the symptoms are often ill-defined. The patient complains of a gradual loss of power, not amounting to paralysis, in one or both lower extremities, which has a tendency to become aggravated and to spread to the upper limbs. Sometimes there are remissions or intermissions in this symptom, and the paresis is distinguished from other spinal complaints by there being no affection of sensibility, nor of the bladder and the rectum. The limbs feel heavy, are difficult to move, and ultimately refuse service altogether. This state is, contrary to ataxy, the same whether the eyes are open or closed. The muscles, however, do not waste, and retain to the last their galvanic and faradic contractility. The absence of pain and other affections of sensibility is, no doubt, owing to the fact that multiple sclerosis affects as a rule the antero-lateral columns of the cord, and therefore leads more to paresis or paralysis, with subsequent contraction, than to anæsthesia or neuralgia; we cannot, however, be certain that we have really to deal with Charcot's disease, until other more characteristic symptoms present themselves, and these are, 1st, a peculiar

kind of tremor, and 2nd, certain signs pointing to an intra-cranial lesion.

The tremor which is peculiar to this affection only becomes manifest when purposive movements of a certain extent are made, and ceases completely during rest. Restricted movements are possible without tremor. This peculiarity distinguishes it from the tremor of shaking palsy, which continues during rest as well as motion, as long as the patient is awake; and also from choreic movements which are disorderly and without purpose, while Charcot's tremor is rhythmic. Thus for instance, when a patient affected with chorea carries a cup of tea to his mouth, we notice at once movements of an entirely contradictory character, which have the tendency to counteract rather than to assist the intended movement; while in Charcot's disease the general direction of the movement persists, in spite of the impediments which are occasioned by the tremor. Again, in progressive locomotor ataxy there may be movements which are devoid of co-ordination, when the arms and hands are affected, and which in a measure resemble choreic and sclerosic movements; but in the movements of ataxy there is no real tremor or oscillations, and they are at fault by being too abrupt and extensive, and therefore devoid of order. The want of co-ordination in the atactic patient is always increased when he closes his eyes, which is not the case in Charcot's disease.

Sclerosic tremor affects not only the upper extremities, but also the head, body, and lower limbs, as soon as purposive movements are made, or when these parts assume a position which can only be maintained by an active contraction of certain sets of muscles. It has, however, been absent in a few exceptional cases, and disappears habitually at a later period of the complaint, when permanent muscular contractions have become established. The tremor is probably owing to the circumstance that the nervous influence is in such patients only transmitted by the cylinder axis, which is deprived of its sheath of myeline, owing to which there is no continuous action, but jerky and irregular oscillations. The persistence of the cylinder axis likewise explains the long duration of the disease, and the circumstance that there is rather paresis than paralysis.

Double vision from paralysis of certain ocular muscles is

now and then observed in the commencement, but is equally transitory as it is found to be in ataxy. Amblyopia is more frequent and permanent, but rarely merges into amaurosis. The ophthalmoscope shows sometimes negative signs, while in other cases there is partial or total atrophy of the optic disc, with extreme attenuation of the bloodvessels. Nystagmus may also be present, and is particularly noticed when the patient endeavours to fix an object with the eyes. The speech is drawling and slow, resembling that which is occasionally found in paralysis agitans; so that a syllable takes nearly a minute before it is pronounced, and there are long intervals between the several syllables. The tongue may be tremulous when protruded; and the symptoms of bulbar paralysis, more particularly difficulty of deglutition and dyspnœa, become sometimes associated with the complaint, and may carry off the patient somewhat suddenly. Where this occurs, we may expect to find patches of disseminated sclerosis in the medulla oblongata and the pons.

A peculiar form of vertigo is frequently observed. All objects seem to the patient to be spinning round, and he himself with them, so that in order to save himself he takes hold of anything near him. The face has a stupid expression; the mouth is open; the saliva is apt to trickle down the chin. The mental faculties, more particularly the memory, are much impaired, and there is great indifference to things in general. Sometimes gastric disturbances such as retching, vomiting, total loss of appetite, are the initial symptoms, and are more or less quickly succeeded by the symptoms peculiar to sclerosis.

The second period of the disease is characterised by aggravation of the symptoms of the first, and the appearance of spasmodic contraction of the extremities, with or without 'spinal epilepsy.' The patient, who until then has still been able to walk about, although with difficulty, is now reduced to the condition of a confirmed invalid. This rigidity of the limbs appears at first only temporarily, under the influence of excitement and other temporary causes. Such attacks may last from a few hours to a few days, and are often separated by considerable intervals, but ultimately a permanent contraction of the limbs is established. They are in a state of extension; the knees are so closely pressed against one another that it is diffi-

cult to separate them, and the feet show the condition of varo-equinus. If the foot is forcibly stretched towards the leg, tremor supervenes at first in the limb acted upon, but may afterwards affect the whole body, and even communicate itself to the couch of the patient; this tremor may last a considerable time, but may be at once arrested by forcible flexion of the big toe (spinal epilepsy). Such a manœuvre also overcomes for the time being the rigidity of the limbs, which become perfectly flexible and relaxed.

In the third period all the organic functions begin to suffer. There is more or less complete anorexia, diarrhœa, and general emaciation. The mind becomes confused, and speech unintelligible, the patient being only able to grunt. Apoplectiform and epileptiform seizures are apt to make their appearance, and are ushered in by absence or complete coma. Hemiplegia is then produced, the limbs being either relaxed or rigid. Death may follow in a few days, and is generally preceded by acute decubitus of the sacrum; but sometimes the patient recovers his consciousness, and the hemiplegia disappears in a short time. Attacks of this kind, however, always leave him more helpless than he was before. During the attacks the pulse is accelerated and the temperature rises to 103° and 104°. If it becomes higher than 104°, a fatal issue may be expected; but where it is less, the mercury often falls suddenly to the normal standard, with simultaneous improvement in all other respects. The apoplectiform attack of Charcot's disease may therefore be distinguished from the apoplectic attack of cerebral hæmorrhage or softening, by the absence of the initial fall of temperature, which is noticed in the latter (p. 102). Complete marasmus is the ultimate result. Paralysis of the sphincters supervenes; there are the symptoms of cystitis and decubitus, with blood-poisoning, or death takes place by intercurrent diseases, such as pneumonia, phthisis, and dysentery. Cerebro-spinal sclerosis generally lasts from six to ten years, while purely spinal sclerosis may spread over twenty years and even more.

5. *Hypertrophy of the brain.*

The weight of the brain varies considerably in different persons, and we have to distinguish between a large and heavy

brain, which is an anatomical and physiological peculiarity, and consistent with perfect health, and an hypertrophied brain, which is disproportionately large to the intracranial cavity in which it is enclosed. The intracranial space may be reduced by hyperæmia, serous effusions, tumours, etc., and these conditions have to be excluded before we can truly speak of hypertrophied brain. In this latter condition there is no excessive development of the cerebral matter itself, which on the contrary is generally found in a state of degeneration, but there is excessive formation of the neuroglia or interstitial connective tissue.

The hypertrophy is generally found in the hemispheres, and also in the medulla oblongata and the pons, while the cerebellum is not liable to it. After the skull-cap has been removed, and the dura mater opened, the brain is seen to surge forward and to project beyond the edges of the bones. The convolutions are compressed and flat, the fissures indistinct, and there is a disproportion in the size of the hemispheres and the cerebellum. The ventricles appear narrow, their walls being pressed against each other, and there is hardly any serum in them. The cerebral tissue is pale, anæmic, dry, tough, and leathery; its membranes are thin and anæmic, and the pia mater is very dry. The skull-bones are thin, their internal surface appears rough, and is sometimes softened. Where the brain becomes hypertrophied in infants, the skull-bones yield to the pressure, and the cavity expands so much that at first sight hydrocephalus is believed to exist, and tapping has occasionally been suggested. In infants this condition is often connected with a strumous dyscrasia, and accompanied with swelling of the lymphatic glands, the thyroid body and the thymus, and a rickety condition of the chest and extremities. It also occurs in middle age, particularly in males, and is then owing to alcoholism, repeated attacks of active and passive cerebral hyperæmia, and depressing mental emotions.

Symptoms of disease come only under observation when the increased mass in the skull-cavity produces pressure, or when incidental circumstances cause irritation, or changes in the quantity of blood contained in the head. In children, however, where the skull is yielding, there is occasionally no distur-

bance of the mental functions, nor of motion and sensation, even when the brain is considerably hypertrophied. The case of a boy is on record who had an enormous head, but was otherwise quite well, except that he often fell when running, as the head fell forward and displaced the centre of gravity of the body. He died of an affection of the bowels, and the brain was found to be extremely hypertrophied. In adults, where the skull is rigid, symptoms of pressure on the brain are the rule, and first affect the power of motion. There is general muscular weakness, more particularly in the lower extremities. The gait is tottering, the patient stumbles easily, and cannot grasp anything with force, or lift anything heavy. Sometimes general paralysis is the result, and followed by convulsions, which are at first rare, partial, of short duration, and mild; but increase as time goes on, and at last merge into epilepsy and eclampsia, probably from the anæmia of the brain, which is habitual in this affection, being suddenly increased by incidental circumstances.

Sensibility is not so much affected. There may, however, be headache, vertigo, tinnitus aurium, photophobia and dilatation of the pupils, and later on dulness of the sentient and sensorial nerves, but no actual anæsthesia. The mind is generally unhinged, there being either excitement, with delirium and mania, or what is more frequent, depression, stupor and idiocy. Vomiting and an extremely slow pulse are also sometimes observed.

The progress of the disease is extremely slow. Where it occurs in children, death is generally owing to incidental diseases, which cause cerebral hyperæmia, and prove fatal from excess of intracranial pressure. When, in adults, convulsive seizures have appeared, the malady runs occasionally a very rapid course, death being comparatively sudden.

6. *Atrophy of the Brain.*

Simple wasting of the brain without any particular degeneration may be congenital, and children subject to it are affected with idiocy, epilepsy, and paralysis. As a rule the atrophy is confined to one hemisphere, most frequently the left, which

ha- only about half the size as, or even less than, the right. The central ganglia appear shrunk and withered, and the aplasia extends to the cerebral peduncle, and the corresponding pyramid, and antero-lateral column of the spinal cord. The ventricles are in a state of extreme dilatation, and contain a large quantity of serum, which is also abundant in the subarachnoid space. The brain-matter appears hard and tough, but is occasionally soft, fragile, and discoloured. This imperfect development of the brain is sometimes owing to premature synostosis of the skull-bones, whereby the intracranial cavity is reduced in size; and the bones are then generally found thickened on the side corresponding to the aplasia. In other instances it arises from congenital hydrocephalus, from extravasation of blood into the cerebral tissue, or from injury and inflammation.

Children who are born with an ill-developed brain, or suffer early in life from consecutive atrophy, lead a wretched and idiotic existence. Where the affection is congenital, or has come on soon after birth, the symptoms are more severe than where a few years have been passed in a physiological condition. The temper is sometimes vicious, the intellect at a low ebb. There is anæsthesia of the special senses, more particularly anosmia and amaurosis, and sometimes deaf- and dumbness. The limbs are paralysed and anæsthetic, but the paralysis is rarely complete, and often accompanied with contraction of the flexor muscles. This form of hemiplegia is also, in contradistinction to others, accompanied with wasting of the paralysed parts. The limbs are short and thin, while the body may appear like that of an adult. It is not only the muscles, but also the bones which are in a state of atrophy, while the skin is normal and there is often a great development of adipose tissue. There is also unilateral atrophy of the face, including the ear, and the symptoms are observed on the side opposite to the affected hemisphere. The bodily functions are otherwise carried on in a regular manner, and the poor idiot may reach a great age. Females menstruate and conceive, but where the aplasia is excessive, the patient generally dies early from incidental diseases.

In exceptional cases one hemisphere has been found wasted, and yet there has been a fair development of the intellectual

faculties during life. This may be explained by assuming a compensatory development of the other hemisphere; and it has generally been found connected with a small quantity of serum in the ventricles and subarachnoid space.

Atrophy of the brain is likewise observed at any time after the organ has reached its full development, and may then be partial or general. Where it is partial it is owing to local pathological conditions, such as softening, hæmorrhage, or encephalitis, and affects all the tissues. Pressure by tumours has the same effect; and where these grow slowly and eventually reach a considerable size, the corresponding atrophy of the tissues, and more particularly the nerve-cells, may be very extensive. Such partial atrophy is confined to one hemisphere, and may affect any part of it. There are often the residues of clots, such as cysts filled with serum or fibrous plates, and the brain-tissue appears withered. The wasting may be stationary or progressive; the latter is chiefly the case in the aged, or where there are several areas of the primary disease, as after repeated attacks of cerebral hæmorrhage, where the wasting spreads from the central ganglia through the pedunculi and pyramids to the lateral columns of the cord. The ultimate products of the disease are heaps of granular cells, and amylaceous and colloid corpuscles.

In such cases the first symptoms are generally those of apoplexy, encephalitis, etc., viz., unilateral and more or less stationary motor paralysis. There is little or no affection of the mental faculties, and of the sensorial and sentient nerves. Matters remain for some time in this state, but when the secondary atrophy progresses, there is gradual deterioration of all functions of the nervous system, and more particularly of the intellectual faculties.

Where cerebral atrophy is general, it is symmetrical and progressive. In such cases chronic alcoholism, attended with the atrophic form of Bright's disease, meningitis, or some general cachexia, such as chronic poisoning by lead and opium, are at the bottom of the complaint. The chief seat of the morbid process is in the neuroglia, and the nerve-cells are only secondarily affected. It occurs mostly in the aged, but cannot be looked upon as mere senile decay, there being actual disease,

Both hemispheres are equally affected. On removing the skull-cap and opening the dura mater, the surface of the brain is found, as it were, at a distance. The convolutions are thin and narrow, the fissures small, and interspersed with deep lacunæ. There is a large quantity of serum in the meshes of the pia mater, in the subarachnoid space, and the lateral ventricles. This is called *hydrocephalus senilis*, or *ex vacuo*. The membranes are thickened and opaque, and the bloodvessels dilated and tortuous; the cerebral substance is generally hard and tough, and only rarely soft and œdematous.

The symptoms observed in this condition are those of gradual loss of cerebral power. There is rarely excitement, except when there are attacks of hyperæmia owing to other causes. Then there may be garrulous delirium, restlessness at night, alternate laughing and crying, fussiness, and tremor. Otherwise we find paralysis and anæsthesia affecting all the different portions of the brain and cranial nerves. The patient is, as it were, in his second childhood: there is more or less imbecility, an entire want of energy or purpose, senile tremor, a slow and intermittent pulse and paralysis of the sphincters; and he is ultimately carried off by bronchitis, decubitus, dropsy, or exhaustive diarrhœa. Cerebral atrophy in its later stages is therefore seen to resemble very closely the last stage of general paralysis of the insane.

7. *Tumours of the Brain.*

The brain is liable to the invasion of numerous adventitious growths, some of which are peculiar to itself, while others are of the same nature as those found in other organs of the body. Tumours in the brain arise from the same causes which produce them elsewhere. There is considerable hereditary influence, and a greater liability of the male sex than of the female, which latter circumstance is probably owing to men being more exposed to injury, and consuming a larger quantity of alcoholic stimulants than women. Now apart from inherited tendency, injury has the greatest influence in causing a tumour to grow, and alcohol in promoting its development. A fall or blow on the head is the most frequent cause, as even where no

coarse lesions are induced at the time, cerebral circulation is upset and disturbed; and rupture of the finer structural elements of the brain may be induced, which is followed by fatty degeneration of the nerve-cells, and proliferation of the neuroglia.

Virchow[1] has shown that the most important tumour peculiar to the brain is *glioma*, which is owing to proliferation of the cementing substance. It is found in the white matter of the hemispheres, and may become as large as a fist. Its growth is essentially slow, and the symptoms caused by it may go on for years. It is either hard or soft, slightly transparent, and according to the vascular development in it, white or pink. The hard glioma is similar to *fibroma*, and is found to contain only scanty cells, which are generally furnished with several nuclei. The soft glioma has little protoplasma, but contains numerous cells which are mostly small. If the cells increase largely, the growth becomes similar to *sarcoma*; and if the basal substance has the character of mucus, it resembles *myxoma*. Glioma is probably always owing to injury, and as there is no constitutional dyscrasia connected with it, the symptoms are purely local and determined chiefly by the seat of the tumour. They are generally slow in their mode of onset, but where there is much vascularity in the growth, there may eventually be attacks of cerebral hæmorrhage, and we have then the well-known symptoms of apoplexy, which are identical with those observed after ordinary cerebral hæmorrhage from rupture of miliary aneurisms.

Another cerebral tumour peculiar to the organ is *psammoma*, or sandy growth, which likewise arises from proliferation of the neuroglia, but contains in addition to it, deposits of chalk in the shape of loose round granules. Psammoma rarely exceeds the size of a cherry-stone; it arises from the dura mater, is generally found at the base of the brain, and has a white smooth surface. *Melanoma*, or pigmentary tumour, proceeds from the pigmentary cells of the pia mater. *Neuroma* is owing to hyperplasia of the grey matter, and is found on the surface of the convolutions, in the ventricles, and in the white medullary

[1] 'Die krankhaften Geschwülste.' Berlin, 1873.

matter of the brain. It attains the size of a millet-grain or a pea. *Cysts* are rare, and if present, are owing to hydrops of the pineal gland or the septum lucidum. *Cholesteatoma* consists of epidermoid cells concentrically deposited, which have undergone horny or fatty degeneration. It resembles epithelioma in structure, but is a perfectly innocent growth; it has a shining mother-of-pearl-like appearance, and is devoid of blood-vessels. Its seat is at the base of the brain.

Other tumours owe their existence to a peculiar constitutional dyscrasia, and the most important of these are cancer, tubercle, and gumma. The gummatous tumour will be considered in the next section. *Cancer* is generally of the encephaloid variety. It almost invariably takes its rise from the dura mater, and proceeds either from the external surface of that membrane towards the skull-bones, which become rarefied and are eventually laid open, when the well-known characters of *fungus durœ matris* are developed; or it grows from the internal surface of the membrane and invades the structure of the brain. It likes to follow the course of cerebral nerves, such as the olfactory and optic, and proceeds through the foramina of the skull, which are provided for the exit of these nerves. It may thus appear in the orbit, the ethmoid bone, and the spheno-maxillary fossa. Histologically the tumour consists of large cells imbedded in a scanty stroma of blood-vessels and degenerated glia-fibres. Its shape is round or puckered, and its colour varies according to the development of blood-vessels, and the degree of retrogressive metamorphosis, being either white, grey, or pink.

Cancer is distinguished by rapid progress, and the signs of general dyscrasia, and is often accompanied with cancerous deposits in other organs.

Tubercle of the brain is connected with the tubercular diathesis, and is chiefly found in children, where it is apt to select the grey matter of the cerebellum. It generally attains the size of a filbert, has a grey or yellowish colour, and is surrounded by a soft layer of blood-vessels and small miliary granules, or by fibrous connective tissue; and it may, as tubercle in other organs, either soften or undergo calcareous metamorphosis. It is generally associated with tubercle in

other organs; frequently commences after acute diseases, such as measles, and is sometimes multiple.

The general effect of tumours on the brain-substance is that of pressure, which produces local symptoms, but also acts on distant parts, in which it may cause fatty degeneration and atrophy. The structures in the neighbourhood of the growth are generally displaced towards the opposite side; the convolutions are flattened, the cerebral tissue is dry, and its blood-vessels anæmic.

The principal symptoms of tumour of the brain are intense headache, vomiting, and optic neuritis; but to these a number of others may be added, according to its seat, mode of growth, and the constitutional peculiarities of the patient.

The initial signs are general malaise, want of energy, irritability of temper, and drowsiness. The patient is depressed in spirits, inclined to be lachrymose, indifferent to the events of daily life, and wishes to be left alone. The memory is impaired, and the patient finds it difficult to fix his attention on any subject. As the tumour proceeds to grow, these mental symptoms become more marked. Depression progresses to melancholia, and where the island of Reil, and the third left frontal convolution are affected, the speech becomes embarrassed, and aphasia is the ultimate result. Towards the end there is either complete imbecility, or attacks of mania with delusions and hallucinations; and the patient dies comatose.

The nerves of special sense suffer considerably from these adventitious growths. Where the seat of the tumour is at the base of the brain anteriorly, the olfactory nerve may be affected, and anosmia caused; but the optic nerve shows almost invariably symptoms of disease. According to Annuske,[1] with whom Dr. H. Jackson agrees, optic neuritis constantly accompanies cerebral tumour, and takes the foremost place amongst the symptoms of intracranial growths. The ophthalmoscopic signs are therefore of the utmost importance in these cases. Increase of intracranial pressure causes wasting of the nervous structures. The brain, being enclosed in a rigid capsule, cannot well escape any considerable amount of pressure, and the cerebro-spinal

[1] Graefe's Archiv,' vol. xix. part 2, p. 165.

liquid is therefore forced between the internal and external sheath of the optic nerve; and as the veins are likewise compressed by the tumour, the secretion of the cerebro-spinal liquid is increased, and hydrops of the ventricles caused, which intensifies the symptoms. The liquid between the two sheaths of the optic nerve is therefore blocked up; the nerve, more especially where it enters the eyeball, is squeezed, and becomes the seat of an œdematous swelling, by which the layers of the retina are ultimately destroyed. Both nerves are affected, although one often more severely than the other. Where the tumour invades the optic tract and the chiasma, the field of vision becomes limited, and there is unilateral or bilateral hemiopia. The amount of damage done depends upon the quantity of liquid which is present, and the amount of pressure by which it is forced onwards.

Increased intracranial pressure causes the appearance of the choked disc, or ischæmia papillæ (Von Graefe's Stauungspapille). There may also be optic neuritis from propagation of the irritation (descending neuritis), and lastly there may be primary atrophy not consequent upon neuritis. Dr. Clifford Allbutt[1] has made a most painstaking investigation into the different forms of optic changes connected with tumours of the brain according to their different localities; and those who wish for further information on this point, must be referred to his volume, and to the standard treatises on the diseases of the eye.

Double vision and strabismus are likewise of frequent occurrence, and arise from pressure on the third, fourth, or sixth nerve. The pupils are generally dilated; they respond only sluggishly to the influence of light, and occasionally differ in size. The portio dura may also be paralysed.

Deafness is so frequently owing to other causes that it cannot be looked upon as a distinctive symptom of cerebral tumour; but it is not unfrequently produced by pressure of the growth on the portio mollis.

There may be unilateral loss of the sense of taste.

Headache is one of the most characteristic signs of tumour,

[1] 'On the use of the Ophthalmoscope in Diseases of the Nervous System,' etc. London, 1871, p. 140.

more especially where this grows rapidly and approaches the membranes. It varies from a simple feeling of heaviness and fulness, to the most agonising pain, which causes the patient to faint away and lose his consciousness for a long time. There are, however, occasionally remissions. It is mostly frontal, and increased by pressure, percussion, and reflex movements, such as coughing and laughing. Where there is localised pain and anæsthesia in the face, and paresis of the masticatory muscles, the fifth nerve is involved by the disease; and where there is numbness and 'pins and needles' in one upper or lower extremity, there is a tumour on the opposite side of the brain. In tumour of the cerebellum, the muscular sense suffers; the gait is tottering, more especially when the patient closes his eyes; and there is vertigo, with an inclination to fall backwards or forwards.

Epileptiform seizures are a common symptom of cerebral tumour, and may either appear in the form of general convulsions and coma, or as unilateral spasm in certain motor areas, without loss of consciousness. Such attacks are sometimes followed by tremor, which may continue for hours. Hemiplegia is also frequent, and may come on from hæmorrhage in the tumour, or in consequence of retrogressive changes in the central ganglia. Where the tumour occupies the median line, or is multiple, or compresses both hemispheres, there may be paraplegia, which is generally associated with paralysis of the bladder.

Vomiting is rarely absent in cerebral tumour, and is usually accompanied with a slow pulse, showing irritation of the pneumogastric nerve.

According to the seat of the tumour the combination of symptoms differs as follows:—where the convexity suffers, there is intense headache and epileptiform seizures, but no paralysis or anæsthesia. In tumours of the frontal lobe there is frontal headache and anosmia, and symptoms of mental excitement; in tumour of the parietal lobe there is slight unilateral paralysis and anæsthesia; in tumour of the occipital lobes: severe headache, vertigo, and melancholia, but no paralysis. Tumour of the base near the chiasma causes anosmia, hemiopia, headache, anæsthesia of the fifth, paresis of the masticatory muscles,

difficulty of deglutition, which may be at first slight and temporary, like laryngeal spasm, but later on becomes more intense and at last amounts to complete dysphagia. There may be nausea, vomiting, flatulency, and other symptoms of dyspepsia arising from irritation of the same nerve. The voice becomes hoarse or is altogether lost. That the cerebellum suffers is shown by attacks of vertigo, unsteadiness of gait, tremor, spasms, numbness, and incomplete paralysis of the lower extremities. Death ultimately ensues from rupture of the aneurism, and is, as usual, preceded by coma and paralysis.

A different group of symptoms presents itself in cases where the *posterior communicating artery*, which establishes a communication between the carotidean and vertebral system of bloodvessels, is affected by aneurism. Here we have at first ptosis, showing that the third nerve, or motor oculi, is compressed. As the tumour grows, other cerebral nerves in the neighbourhood become affected; there is strabismus, an immovable and dilated pupil, amblyopia and amaurosis with choked disc. Headache, vertigo, drowsiness, stupor, and certain forms of insanity come on at a later period, showing that the nutrition of the cortical substance of the brain suffers.

8. *Syphilitic affections of the Nervous System.*

Nerve-syphilis is one of the most interesting chapters of pathology, and has only recently been thoroughly investigated, more especially by Professor Waller, of Prague, M. Lanceraux,[1] Dr. Wilks,[2] Dr. Buzzard,[3] Professor Heubner,[4] and Dr. H. Jackson.[5] The practical importance of pathology in this instance is extremely great, since it has been found that the most formidable syphilitic diseases of the nervous system, such as epilepsy and paralysis, are much more readily and thoroughly curable than the corresponding idiopathic maladies, which do not owe their origin to this subtle poison.

[1] 'Traité historique et pratique de la syphilis.' Paris, 1866.

[2] 'Guy's Hospital Reports,' 1864 *seqq.*

[3] 'Clinical Aspects of Syphilitic Nervous Affections.' London, 1874.

[4] 'Die luetische Erkrankung der Gehirnarterien,' Leipzig, 1874; and in Ziemssen's 'Handbuch,' &c., vol. xii., 1877.

[5] 'Journal of Mental Science,' July, 1875.

Neuro-syphilitic affections mostly belong to the latest period of the constitutional disease, viz., the so-called tertiary stage, or to the later portions of the secondary stage. They are invariably preceded by a hard infecting sore, and generally by secondary symptoms affecting the skin and fauces; but in some instances they appear as the first manifestations of constitutional syphilis, and that in from twelve months to twenty years from the appearance of the primary affection. In a few exceptional cases they have closely followed upon the first rash and sore-throat. They may occur at all periods of life, in children as well as in old persons, but are most frequent between twenty and forty years of age, and this circumstance is of diagnostic importance, as hemiplegia or paraplegia coming on in youthful persons is in nine cases out of ten of syphilitic origin. The male sex is more liable to them than the female, which is in accordance with the fact that constitutional syphilis is altogether more frequent in men than in women.

Nerve-syphilis appears to occur chiefly in those persons in whom there is evidence of the neuro-pathic constitution, either hereditary or acquired. There is almost always a family history of apoplexy, epilepsy, chorea, megrim, or other nervous maladies, and frequently the patient himself has previously suffered from neuralgia or fits. Persons who have put an undue strain on their nervous power, either by excessive mental labour, or by free indulgence in alcohol and the sexual appetite, are more liable, when rendered syphilitic, to become subject to nervous maladies, than those in whom there have been no such antecedents. Injury, such as a blow on the head, or a fall, and depressing emotions, act frequently as exciting causes of these diseases. Finally, an unsystematic and too soon interrupted treatment of the primary affection, has to be looked upon as a powerfully predisposing cause of nerve-syphilis.

Syphilis affects with preference the brain and cerebral nerves, but does not spare the spinal cord. Anatomically we find that the characteristic lesions are not meningitis or encephalitis, as was formerly believed, but repeated attacks of hyperæmia, tumour, and disease of the arteries.

a. Syphilis of the Brain and Cranial Nerves.

a. The *congestive form of cerebral syphilis* shows hardly any striking features on the post-mortem table, more especially where the case ends fatally at an early stage of the complaint, from such complications as cystitis, decubitus, phthisis or pneumonia. Where the disease has lasted for a considerable time, the membranes of the brain are seen to have lost their transparency, and there is slight wasting of the cerebral convolutions, which latter, however, is not sufficient to explain the severe symptoms which have been observed during life. The lesions are the same, although in a slighter degree, as those which are found in general paralysis of the insane, and affect more particularly Hitzig and Ferrier's psycho-motor centres in the cineritious substance of the anterior lobes. In some of these cases the cervical sympathetic nerve has been found in a state of pigmentary degeneration, and it is probable that disease of the superior cervical ganglion of that nerve may have an important influence in the production of the repeated attacks of hyperæmia, by which this form of brain syphilis is characterised.

The symptoms which are observed under these circumstances, resemble very closely those of general paralysis of the insane. They are at first indefinite, come and go, and a change in the mind and temper is the most characteristic feature. There is excitement or depression, with confusion of thoughts, fussiness, and ambitious ideas or delusions. Apart from a general feeling of malaise, the patient does not complain of being ill. As time goes on there is loss of energy, debility, embarrassed speech, partaking of the nature of aphasia, and being cortical rather than medullary in its kind. The size of the pupil is unequal, the tongue is tremulous when protruded, there is tottering gait, and 'pins and needles' and numbness in the extremities are complained of. When the symptoms on the part of the nervous system become more marked, there is often a simultaneous outbreak of fresh syphilitic manifestations on the skin, mucous membrane, or periosteum. The intellect and memory now suffer more decidedly, and symptoms of

paralysis appear from time to time, viz., aphasia, agraphia, hemiplegia and paraplegia. Such symptoms may last at first only for a few hours or days, but they gradually become more permanent. The general debility increases *pari passu*, and unless an energetic anti-syphilitic treatment be perseveringly followed, the patient dies within a few years from the outbreak of the disease, from cystitis, decubitus, and general marasmus.

β. The second manifestation of cerebral syphilis is the *syphilitic tumour*, *gumma*, or *syphiloma*, which presents itself in two varieties, these being probably only different stages of development of the same deposit. There is the soft and hard variety, the soft tumour being the earlier, and the hard swelling the later phenomenon. The *soft tumour* consists of a reddish-grey jelly, from which on section a small quantity of pinkish liquid is seen to escape. Its histological elements are round cells and nuclei, mixed with spindle and stellated cells, and few, but large capillary vessels. The outline of such tumours is not well defined, and they seem gradually to merge into the surrounding normal tissue. They are chiefly found in the sub-arachnoid space, and grow from there to the surface of the brain; but they also occur in the dura mater, and are in this situation generally harder than when situated in the soft and moist tissue of the pia.

The *hard tumour* is in many respects similar to tubercle. It is dry, yellow, of a cheesy consistency, and on section homogeneous. It occurs interspersed into the reddish-grey jelly, which I have just described, or as a well-defined tumour of variable size. It consists histologically of a granulated substance otherwise devoid of structure; there are no blood-vessels or spindle-cells, but now and then heaps of pigmentary granules and crystals, and oil-globules near the periphery. Its size varies from that of an almond kernel to that of a pigeon's or even a hen's egg, and its shape is frequently adapted to that of the spaces in which it is discovered. It is found between the two layers of the dura mater, which are much thickened, and more especially in the falx cerebri. The skull-bone, which corresponds to its situation, is generally in a state of dry caries, and appears rough and attenuated while the other portions of the skull are normal. The yellow, hard tumour is probably

owing to contraction and atrophy of the soft gumma. It is likewise found in the subarachnoid space, and from there proceeds to the bloodvessels, nerves, and cerebral tissue itself. Occasionally all the membranes and the cineritious substance are grown together into a uniform mass, and cannot be separated, and the two varieties of tumour are then seen together. Soft jelly is embedded between the dura and the surface of the brain, and one or several dry yellow tumours are lying in the fissures between the convolutions. The surrounding brain-tissue is in a state of red or white softening, or a portion of the cortical and medullary matter is changed into the same cheesy mass of which the tumour itself consists. Under the influence of anti-syphilitic treatment, nearly the whole of these changes may be repaired, and cicatricial patches are then discovered on the surface of the hemispheres.

At the base of the brain there is occasionally a diffuse infiltration with grey jelly, which can no longer be called a tumour, but must be looked upon as gummatous meningitis. The effusion in such instances is seen to spread from the olfactory bulb to the posterior portion of the cerebellum, and may even invade the cortex.

The symptoms of cerebral syphiloma differ considerably from those of syphilitic hyperæmia; and one that is hardly ever wanting, and also the first to appear, is a peculiar kind of headache, which appears chiefly at night and is relieved towards morning. It is intolerably severe, and occurs in paroxysms which last for a few weeks, after which there is a remission, which is again succeeded by a fresh outbreak of it; and unless specific treatment be adopted, this may go on for years. The seat of the headache is mostly at the sides of the head, but it may also be frontal and occipital. It is sometimes localised in a very small area, and then generally increased by pressure; and it is owing to a gummatous deposit on the internal surface of the skull-bones, which irritates the periosteum and the dura mater. Sleeplessness is another symptom, which is generally caused by the pain, but also occurs during free intervals; and it is apt to raise our suspicions, because it is mostly found in young persons, in whom insomnia is otherwise very rare.

After these symptoms have continued for a variable time,

epileptiform attacks are apt to supervene, which sometimes resemble in every way the ordinary attacks of primary epilepsy.

Unilateral convulsion without loss of consciousness is often connected with this condition; the muscular spasm starts from the thumb or first finger, or the foot, or the face, and affects only the arm, or the leg of the same side, but becomes bilateral where the nerve nuclei of the two sides are associated; or there may be a regular epileptic seizure, *i.e.*, general convulsions and coma. When the hemispasm is on the right side, showing affection of the left hemisphere, temporary aphasia may be produced, and there may be hemiplegia with this, or left hemiplegia without aphasia. This hemispasm is due to irritation of the convolutions of the opposite hemisphere. When such fits succeed each other more or less rapidly, the mind becomes affected. There is irritability of temper; the patient is sometimes in a state of hysteria, laughing and crying alternately without any adequate cause; he is generally depressed in spirits; the memory is impaired, and the current of thoughts considerably retarded. The speech is embarrassed; the patient is unable to finish a sentence, and sometimes stops for a minute without being able to go on. He tries to help himself with gestures, but even this aid after a time forsakes him; and complete aphasia, agraphia and amimia may become developed.

In such cases there is no hemiplegia or any other form of actual paralysis, but there is paresis such as we are apt to connect with disease of Hitzig's and Ferrier's psycho-motor centres, rather than with an affection of the central ganglia. The patient is still able to walk, dress, and feed himself; but the gait is unsteady, the foot drags on the ground, and there is ataxy in the movements of the hands and fingers. Even at this stage he may completely recover, if an energetic treatment is directed to the cause of the disease; but without such interference the symptoms gradually become worse. There are frequent epileptic fits, sometimes developing into epilepticism (p. 241) with coma, from which the patient does not awake; or decubitus is developed, which leads to blood-poisoning; or death supervenes from total exhaustion of the nervous force.

The symptoms just described arise from syphiloma of the sub-arachnoid space, which gradually involves the cortex of the

brain and the adjacent medullary matter. Where the third left frontal convolution and its neighbourhood is suffering, aphasia and agraphia will be the result; where the anterior lobes are affected there will be symptoms of paresis; while irritation of the posterior lobes causes melancholia without much, if any, loss of motor power. But whatever portion of the cineritious structure is affected, syphilitic epilepsy will be the most prominent symptom.

The cerebral nerves generally suffer in exact proportion to the seat of the syphiloma, which causes irritation and finally destruction of the nerve-trunk. The gummatous tumour sometimes grows right round a nerve and compresses it, or it squeezes it against the bone, or an exostosis may occur in the osseous canal through which the nerve has to pass. It is quite true that a nerve is occasionally discovered passing right through a syphiloma at the base without having lost its function or structure, but such cases are exceptional.

The general sequence of events is neuritis followed by atrophy. The nerve appears at first reddish and softened, its sheath is thickened, and at a later period it is wasted and changed into a thin thread. Sometimes the sheath of the nerve appears perfectly normal, but on opening it the nervous substance is seen to have disappeared and to be replaced by a reddish or yellow mass, corresponding in structure to the soft and hard syphiloma. In other instances the syphiloma grows directly from the pia mater along the bloodvessels into the nervous substance, more particularly into the chiasma of the optic nerves, causing atrophy of the same. Finally there may be no structural lesion, although symptoms of paralysis, anæsthesia and neuralgia may have been present during life. Dr. H. Jackson [1] is therefore incorrect in stating that the pathogenesis of these cases is nothing but 'squeezing of nerve-fibres by overgrowth of the connective tissue element of the nerve-trunk.' This is only one of several causes.

Amongst the cranial nerves the third is most frequently affected by syphilis; and the most common symptoms are ptosis, external strabismus and paralytic dilatation of the pupil. Vertigo is occasionally a symptom of paralysis of this nerve.

[1] 'Journal of Mental Science,' July, 1875, p. 5.

Syphilitic neuro-retinitis and simple retinitis are also common. There is effusion of serum into the layers of the retina, which is sometimes slight and sometimes considerable, and is generally preceded by hyperæmia. The outline of the disc is rendered indistinct and hazy, and the neighbourhood of the yellow spot is particularly affected. Sometimes the vitreous humour becomes turbid and prevents a thorough ophthalmoscopic examination. Where the effusion takes place rapidly, the sight may be quickly destroyed; but in most cases it occurs slowly. It is not unfrequently associated with irido-choroiditis. Dr. Jackson has pointed out that double optic neuritis may often be recognised by the ophthalmoscope before vision suffers, and that it may be for some time the only symptom, or be accompanied with such slight symptoms as to be hardly noticed.

The portio dura and the fifth and sixth nerve may likewise suffer; there is then inability to close the eye, neuralgia of the face, with lachrymation, paralysis of mastication, and internal strabismus.

γ. The third and last form of cerebral syphilis is *disease of the arteries*, which affects with preference the carotids, the circle of Willis, the sylvian artery, and that of the corpus callosum. The first symptom of disease is, that the artery becomes less transparent, and loses its pink colour, assuming instead of it a white greyish appearance. At the same time the vessel loses its cylindrical shape and becomes quite round, while its coat is hardened and gives a cartilaginous sensation to the finger. The diameter of the vessel is very much reduced by the deposition of a moist gray substance, which later on becomes hard and dry; and what remains of a free canal is often blocked up by thrombosis, so that ultimately the whole artery is changed into a solid cord. This deposit takes place chiefly between the endothelium and the elastic fibres of the vessel. At first it appears to consist of endothelial cells, which multiply considerably and develope into connective tissue. This growth goes on in a longitudinal as well as in a transverse direction, and the degeneration is therefore apt to spread to the branches of the artery.

I have already spoken of the two spheres of cerebral nutrition of Heubner and Cohnheim (p. 148), viz., of the basal and

cortical sphere. In syphilitic disease of the cerebral arteries this distinction becomes of paramount importance. The basal sphere comprises the vertebral, basilar, and carotid arteries, the circle of Willis, and the commencement of the anterior, middle, and posterior cerebral arteries. All these vessels give off small branches vertically, which penetrate directly into the cerebral matter, become divided and terminal, and then proceed through the capillary vessels into the smaller veins. It is particularly in this basal sphere, which supplies the central ganglia, that plugging of the arteries from deposit and subsequent thrombosis becomes so dangerous to the nutrition of the parts; for as there is no anastomosis, the various forms of necrobiosis, such as red, yellow, and white softening, are easily produced, the result being generally syphilitic hemiplegia.

In the cortical sphere of nutrition, on the other hand, the plugging of arteries is not of the same vital importance, because the peripheral part of the bloodvessel may still be supplied with blood by anastomosis in the pia mater. The cortical arteries run for a long time in the pia mater without giving off any branches to the cerebral substance; they divide in the pia, become constantly smaller, and anastomose so thoroughly with their fellows that a kind of network is established, by means of which not only the smaller branches but also the principal arteries are made to communicate with each other. The cerebral matter only receives small capillary vessels from this large vascular net after it has been allowed to spread over a considerable surface. Although, therefore, the danger of starvation is much less in the cortex than in the central ganglia, nevertheless, a rapid plugging cannot pass without causing mischief, inasmuch as it decreases the pressure in those vessels; while if collateral circulation is established, the pressure may be suddenly increased above the normal standard. The cineritious substance is thus exposed to considerable vicissitudes of circulation, and the temporary apoplectic seizures, which are so common in this form of syphilis, find in them a satisfactory explanation.

In both spheres of nutrition, however, the basal as well as the cortical, simple narrowing of the arterial tubes, without actual plugging of the same, must have a deleterious influence on the nutrition of the entire brain. It increases the resistance

offered to the current of blood, which becomes further retarded by the rigidity of the tube, which has lost its elasticity. The interchange of oxygen and nutritive material is therefore considerably lessened, which explains the loss of energy, the impairment of the mental faculties, and the somnolence which is found in a number of these cases.

Where the basal sphere of nutrition suffers, the symptoms are generally rapidly developed. It is not by any means rare that, after a few insignificant premonitory symptoms, there is a sudden stroke of apoplexy which proves fatal. The symptoms of this form of apoplexy are in all respects similar to those which have been described as ordinary apoplexy from cerebral hæmorrhage. Multiple thrombosis of several important basal arteries is discovered *post mortem*.

In other cases there are premonitory symptoms chiefly on the part of the cranial nerves. There is ptosis, double vision, weakness of sight from optic neuritis, anæsthesia, and neuralgia in certain branches of the fifth nerve, spasm in the portio dura and sixth nerve, etc. These symptoms may come on, as it were, spontaneously, or after mental and physical efforts, excitement, and indulgence in alcohol and the sexual appetite. After a time there is a somewhat slowly-produced attack of hemiplegia, with or without aphasia, and without loss of consciousness. If collateral circulation is established the patient may gradually improve; or he sinks into a somnolent condition, resembling that of typhoid fever. There is headache, confusion, fussiness; the patient has a staring, absent look, and a morose expression of the countenance; he sometimes gets out of bed and passes water or fæces in the middle of the room, and does other things which show absence of the feeling of shame; but on being talked to, he generally becomes more reasonable. He will often refuse food, and die in the first or subsequent attacks; yet where all these symptoms have been present, an immense improvement may by proper treatment be brought about in the patient's condition.

Where there has been true hemiplegia, recovery is generally imperfect, even under the best treatment. There are, however, temporary kinds of hemiplegia which only persist for a day or two, and where the starvation of cerebral tissue is evidently of

too short duration to cause any great degree of softening. In some cases hemiplegia is followed by Türck's sclerosis of the lateral columns of the spinal cord, just as after ordinary cerebral hæmorrhage.

b. Syphilitic affections of the Spinal Cord.

These are much more rare than the corresponding diseases of the brain and cranial nerves, and there are as yet only few post-mortem examinations of such cases on record.

The syphiloma occurs in its two forms, viz., as jelly and as cheese, in the pia mater and the subarachnoid space; and the three membranes are grown together with each other and with the surface of the cord. There are, however, not circumscribed tumours as in the brain, but we meet rather with a kind of infiltration of the meninges and lymphatic spaces by gummatous effusions, which appear small, multiple and disseminated. Where the membranes grow together with the periosteum of the vertebræ and the surface of the cord, there is generally proliferation of the neuroglia and wasting of the white columns. Some cases in which the symptoms of acute ascending spinal paralysis are observed during life, seem to be owing to hyperæmia simply, as no structural alterations of the cord have been discovered after death.

In this latter case the symptoms generally commence at an early period, viz., in the first year, and are accompanied by the usual early manifestations of constitutional syphilis. The first symptom is sudden paraplegia, with incontinence of the urine and fæces. There is no pain in the spine, and no anæsthesia of the limbs. Decubitus soon becomes developed, and the patient dies within a few weeks from the beginning of these symptoms.

More frequently, however, paralysis comes on in the later periods of the disease, after many other symptoms have existed for a long time. There is muddy pallor of the skin, and a disagreeable smell about the patient, who is generally feeble and in a state of constant malaise. He experiences pain at different points of the spine, which is increased by pressure; and also pain, 'pins and needles,' numbness and stiffness in the lower extremities. These symptoms come and go, and then there is all of a sudden an attack of paraplegia or hemi-para-

plegia. Where the seat of the disease is in the lower portion of the dorsal cord, there is also paralysis of the sphincters. If the case is not well treated, the paralysis remains stationary, and ultimately decubitus is developed, which shortly leads to a fatal result. By proper treatment, however, the patient may get well in a very short time. Some years ago I was consulted by a patient of this kind only two days after the paraplegia had become developed. He was carried into my consulting-room on the back of a cabman, and had completely lost the power over the lower extremities, but only slightly over the sphincters. There was no anæsthesia. Under full doses of iodide of potassium the patient improved most rapidly, and walked briskly into my room a week after I had first seen him. He did not, however, perfectly recover, as a slight degree of weakness in the left leg has remained up to this day.

Where the cervical spine is affected, matters are more serious. There is then not only paraplegia and paralysis of the sphincters, but also of the thoracic and abdominal muscles, the upper extremities, and the diaphragm. Asphyxia from paralysis of the phrenic nerve, or pneumonia, generally carries the patients off in a short time, unless, as we have seen it, the remedy proves stronger than the disease. But in cases of this class we cannot look forward to perfect recovery, as the posterior columns of the cord generally become disorganised beyond a thorough repair, and a state resembling locomotor ataxy may then remain for life.

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